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CHENNAI, TAMILNADU.

ETIO-PATHOLOGICAL PROFILE OF CHRONIC PERICARDITIS

Dissertation submitted for DM
(Branch II – Cardiology)

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CERTIFICATE

This is to certify that this dissertation titled **"ETIO-PATHOLOGICAL PROFILE OF CHRONIC PERICARDITIS"** submitted by **Dr.S.BALASHANKARGOMATHI,** to the faculty of Cardiology, The Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the requirement for the award of DM degree Branch [Cardiology] is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, Dr.S.Balashankargomathi, solemnly declare that the dissertation titled **"ETIO-PATHOLOGICAL PROFILE OF CHRONIC PERICARDITIS"** has been prepared by me.

This is submitted to **The Tamilnadu Dr.M.G.R.Medical University**, Chennai, in partial fulfillment of the regulations for the award of DM degree Branch [Cardiology].

Madurai.
Date:

Dr.S.BALASHANKARGOMATHI

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INTRODUCTION

A wide variety of conditions may result in pericardial effusion. The etiologic spectrum in different series largely depends on the source of the patients, the characteristics of the centre, and on the frequency distribution of the different etiologies in each geographic area. In many cases, pericardial effusion is associated with a previous known condition or underlying cardiac diseases, which are finally proven to be the cause of the pericardial effusion. In patients with no apparent cause of pericardial effusion, the presence of inflammatory signs are predictive of acute (idiopathic) pericarditis; on the other hand, sever effusion with absence of tamponade is predictive of chronic idiopathic pericardial effusion. Tamponade without inflammatory signs is suspicious for neoplastic pericardial effusion. The prognosis of pericardial effusion is related to the underlying disease, being especially poor in patients with malignancy.

The series by Colombo etal included 25 male patients, all of whom were submitted to an invasive pericardial procedure. Of these patients, 44% presented with cardiac tamponade. The most frequent causes of pericardial effusion were: neoplastic (36%), idiopathic (32%), and uremic (20%).

Corey etal investigated the etiology of pericardial effusion in 57 patients. Etiologic diagnosis was made in 53patients (93%). The most common diagnoses were malignancy (23%), viral infection (14%), radiation induced inflammation (14%), collagen –vascular disease (12%), and uremia (12%). In only four patients there was no diagnosis made.

Sagrista –Sauleda etal included 322 patients, 132 with moderate and 190 with severe pericardial effusion. Cardiac tamponade was present in 37%. In this series, the most common diagnosis was acute idiopathic pericarditis which accounted for 20% of patients. The next most

prevalent diagnoses were iatrogenic effusion (16%), neoplastic effusion (13%), and chronic idiopathic pericardial effusion (9%).

In the 56 patients with tamponade included in the study of Guberman etal, the most common diagnoses were metastatic cancer in 18 patients, idiopathic pericarditis in eight patients, and uremia in five. Once again, the worst prognosis was in the group of patients with cancer.

Study by Levine etal, involving 50 patients, the most frequent etiologies of pericardial effusion were malignancy (58%), idiopathic effusion (14%), and uremia (14%).

In a consecutive series by Blake S etal of 32 cases of chronic constrictive pericarditis four patients were attributable to rheumatoid disease, two patients to trauma, one patient to sarcoidosis, and four patients to tuberculosis. In the remaining 21 cases the etiology was not found. Therefore the main causes of large pericardial effusion in general medical centres are idiopathic pericarditis and malignancy.

CLINICAL CLUES TO ETIOLOGY:

Agner and Gallis, in a retrospective series of 133 patients, observed that hemodynamic compromise, cardiomegaly, pleural effusion, and large pericardial effusion were more common in patients with Tuberculous or malignant disease than in patients with idiopathic pericarditis.

In the series of Posner etal, dealing with 31 patients with cancer and pericardial disease, patients with malignant pericardial disease had tamponade more frequently, whereas fever, a pericardial friction rub, and improvement following treatment with non-steroidal anti-inflammatory drugs characterized patients with idiopathic pericarditis. The predictive value of these different clinical findings for assessing the etiology of pericardial effusion was not established.

Recent prospective study of 322 patients with moderate and severe pericardial effusion, investigated the value of selected clinical, data (underlying disease, development of cardiac tamponade, and presence or absence of inflammatory signs) for inclusion of the patients in a likely major etiologic diagnostic category. In 60% of the patients a known previous condition that could cause pericardial effusion was present. The pericardial effusion was shown to be related to the underlying disease in all but seven of these patients. In the patients with no apparent cause of pericardial effusion, the presence of inflammatory signs (characteristic chest pain, pericardial friction rub, fever or typical electrocardiography changes) were predictive for acute idiopathic pericarditis ($p<0.001$, likelihood ratio 5.4), irrespective of the size of effusion and presence or absence of tamponade. Furthermore, a large effusion with absence of inflammatory signs and absence of tamponade was predictive of chronic idiopathic pericardial effusion ($p<0.001$, likelihood ratio 20), and tamponade without inflammatory signs was predictive of neoplastic pericardial effusion ($p<0.001$, likelihood ratio 2.9).

The diagnosis can be established through general examination, including the search of tubercle bacilli in sputum or gastric aspirate (which provided the diagnosis in four of our eight cases), or by means of pericardial fluid or pericardial tissue examination (indicated in patients pericarditis with tamponade or with persistent active illness for more than three weeks).

Echocardiographic clues to etiology:

Band-like intrapericardial echoes and masses were initially described in the pericardial cavity in patients with pericardial effusion of varying etiology. In fact most of such patients were associated with malignancy.

The intrapericardial abnormalities associated with tuberculous pericardial effusion were

reported by Chia etal. He described linear frond-like echo dense structures protruding into the pericardial cavity forming a dense mass as a result of Fibrinous pericarditis.

Liu etal, demonstrated that thickened pericardium and fibrin strands were highly specific (94% and 88% respectively), and exudative coating had a high sensitivity (100%), but low specificity (22%), in the diagnosis of tuberculous pericarditis. The thickness of the exudative coating/deposits by echocardiography was found to be 4.5 (2.0) mm in tuberculous pericardial effusion and 2.8 (0.69) mm in chronic idiopathic pericardial effusion ($p<0.013$). Intrapericardial echo abnormalities such as a greater degree of pericardial thickening, and thickness of exudative coating or deposits, and strands crossing the pericardial space, are useful in the diagnosis of tuberculous pericardial effusion and in differentiating this entity from chronic idiopathic pericardial effusion.

As there are wide variations in etiology of chronic pericardial diseases, we did a research mainly to findout the etiological profile of chronic pericardial disease. This will be helpful in deciding the long term therapy in those patients. This also has prognostic implications.

REVIEW OF LITERATURE

Introduction:

Pericardium is specialized structure to serve its complex active and passive functions. The pericardium is composed of the parietal pericardium (an outer fibrous layer) and the visceral pericardium (an inner serous membrane made of a single layer of mesothelial cells). The visceral pericardium is attached to the epicardial fat and reflects back on itself to form the parietal pericardium. The pericardium normally contains as much as 50 mL of an ultrafiltrate of plasma. Drainage occurs via the thoracic duct and the right lymphatic duct into the right pleural space.

Pericardial physiology includes three main functions. First, through its mechanical function, the pericardium promotes cardiac efficiency by limiting acute dilation, maintaining ventricular compliance with preservation of the Starling curve, and distributing hydrostatic forces. The pericardium also creates a closed chamber with sub atmospheric pressure that aids atrial filling and lowers transmural cardiac pressures. Second, through its membranous function, the pericardium shields the heart by reducing external friction and acting as a barrier against extension of infection and malignancy. Third, through its ligamentous function, the pericardium anatomically fixes the heart.

It contains two types of sinuses. The transverse sinus lies in front of the atria and superior venae cavae and behind the proximal ascending aorta and pulmonary artery as a short pericardial passage. The oblique sinus forms a separate recess with the pulmonary veins and forms an inverted "U", framing a posterior projection. CT and MRI disclose numerous smaller recesses. The pericardial sinuses and recesses increase the pericardial capacity to accommodate

increased fluid (or other contents), contributing to the pericardial reserve volume.

Virtually pericardium is involved in various diseases. Pericardial diseases have various clinical patterns.

Clinical pericardial syndromes are:

- ❖ Acute pericarditis
- ❖ Relapsing pericarditis
- ❖ CardiacTamponade
- ❖ Chronic pericardial effusion without compression
- ❖ Effusive constrictive pericarditis
- ❖ Constrictive pericarditis

Some important causes of pericarditis:

Idiopathic

Infectious

Viral

Bacterial

Fungal

Parasitic

Immunological

Relapsing pericarditis

Post infarction (Dressler's syndrome)

Post cardiotomy syndrome

Rheumatic fever

Still’s disease

Rheumatoid arthritis

Systemic lupus erythematosus (SLE)

Mixed connective tissue disease

Polyarteritis and the Churg-Strauss variant

Neoplastic

Post-irradiation

Traumatic

Uremic

Drug induced

Patterns of pericardial diseases

Viral pericarditis

Viral pericarditis is the most common infectious disease of the pericardium. It is caused by either direct viral attack, the immune response following the viral infection, or both.

The following viruses are usually responsible for pericarditis.

- Enterovirus,
- Adenovirus,
- Cytomegalovirus (CMV),
- Ebstein-barr virus,
- Herpes simplex virus,
- Influenza virus,
- Parvo b19 virus,
- Hepatitis C virus,

- HIV.

Initial presentation often resolves within two weeks. In up to 50% of patients it may recur. It may present with acute effusive pericarditis; tamponade, constriction and "dry" pericarditis. Arrhythmias or conduction defects indicate the presence of associated myocarditis or other concomitant heart disease. Evaluation of pericardial effusion and/or pericardial/epicardial tissue by polymerase chain reaction (PCR) or in situ hybridization may be helpful in arriving the etiology. A fourfold rise in serum antiviral antibodies is suggestive but not diagnostic.

In chronic or recurrent symptomatic pericardial effusion due to viral infection, the following specific treatment is under investigation

(1) **CMV pericarditis:**

- Hyper immunoglobulin once daily,
- 4ml/kg intravenously on days 0, 4 and 8;
- 2ml/kg on day 12 and 16

(2) **Coxsackie B pericarditis**

- Interferon alpha $2.5x10^6$ IU/m^2 subcutaneously three times a week

(3) **Adenovirus and Parvo virus B19 positive Myocarditis**

- Hyper immunoglobulin 10g IV on days 1and 3

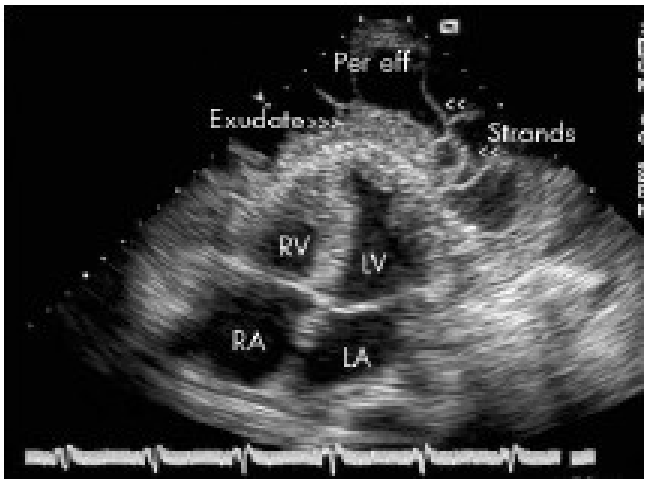
Auto reactive pericarditis

It is characterized by recurrent episodes of pericardial effusion. It has following specific features. Pericardial fluid analysis revealed an increased number of lymphocytes/mononuclear

cells >5000/mm^3 (auto reactive lymphocytes), or the presence of antibodies against heart muscle tissue in the pericardial fluid (auto reactive antibody mediated); (2) signs of myocarditis on epicardial/endomyocardial biopsies (≥14cells/mm^2); (3) exclusion of active viral infection in pericardial effusion and endomyocardial/ epimyocardial biopsies (no virus isolation, no immunoglobulin IgM titre against cardio tropic viruses in pericardial effusion, and negative PCR for major cardio tropic viruses); (4) tuberculosis, Borrelia burgdorferi, Chlamydia pneumoniae, and other bacterial infection excluded by PCR and/or cultures; (5) no neoplastic infiltration in pericardial effusion or tissue; and (6) exclusion of uremia, systemic, and metabolic disorders.

Tuberculous pericarditis:

Tuberculous pericarditis can present with various clinical manifestations. It may present with acute pericarditis; chronic pericardial effusion without compression; Effusive-constrictive pericarditis; Relapsing pericarditis; Cardiac Tamponade and Constrictive pericarditis. Pericardial constriction occurs in 30-50% of cases and mortality in untreated disease approaches 85%. The absolute criteria for diagnosis are the identification of mycobacterium tuberculosis in the pericardial fluid or tissue, and/or the presence of caseousgranulomas in the pericardium. Sputum cultures, ELISPOT skin test, analyses of pericardial effusion by acid-fast staining, mycobacterium culture or radiometric growth detection (for example, BACTEC -460), adenosinedeaminase (ADA) , pericardial lysozme, and PCR for M tuberculosis are various modalities has been used for the diagnosis of Tuberculous pericarditis. High ADA (> 40U/I) in pericardial effusion is diagnostic for Tuberculous pericarditis (93% sensitivity, 97% specificity) and it is a prognostic indicator for pericardial constriction. “PCR is as sensitive (75% v 83%), but more specific (100% v 78%) for Tuberculous pericarditis than ADA.



Tuberculous pericarditis should be promptly treated with a combination of three or four tuberculostatic drugs for 9-12 months. In a study by Gooi HC etal forty-one patients with acute tuberculous pericarditis were studied retrospectively. Anti-tuberculosis chemotherapy alone was effective in thirty patients. Five patients died, two from unrelated causes, two due to delayed diagnosis, and one after pericardiectomy. Constrictive pericarditis developed in seven patients, six of whom had successful pericardiectomy. Corticosteroids could not be shown to have reduced the risk of developing constriction. When constriction occurred it did so within the first six months of illness in all cases. Prednisone should be administered along with antituberculous drugs in relatively high initial doses (1mg/kg) and maintained for 5-7 days. It should be progressively reduced over the span of 6-8 weeks. Corticosteroids reduce the host reaction to mycobacterial infections, minimize exudation, fibrin deposition, and proliferation of tuberculomata, may decrease symptoms and signs, and reduce mortality in Tuberculous pericarditis. If symptoms and sings of constrictive pericarditis remain 6-8 weeks after full tuberculostatic and corticosteroid treatment, Pericardiectomy is indicated.

Fungal pericarditis

Fungal pericarditis occurs mainly in immunocompromised patients (Candida, Aspergillus, Blastomyces, Nocardia, and Actinomyces species) or in the course of endemic fungal infections (Histoplasma, coccidioides).The clinical picture comprises the full spectrum of pericardial diseases including fungal myocarditis. Diagnosis is obtained by staining and culturing pericardial fluid or tissue, and antifungal antibodies in serum are also helpful.

Pericarditis in renal failure:

Uremic pericarditis

It is the Fibrinous inflammation with adhesions between the thickened pericardial membranes ("bread and butter" appearance) caused by the high degree of azotemia (blood urea nitrogen usually> 60mg/dl) in advanced renal failure before dialysis or shortly thereafter.

Dialysis associated pericarditis

It occurs in patients on maintenance haemodialysis, and occasionally with chronic peritoneal dialysis. It is caused by inadequate dialysis and /or fluid overload. After renal transplantation pericarditis may be caused by uremia or infections (for example, CMV)

Most patients with uremic pericarditis respond within 1-2 weeks to haemo-or peritoneal dialysis. Heparin-free haemodialysis should be used to avoid intrapericardial hemorrhage. Patients with pretamponade should undergo pericardiocentesis before haemodialysis since acute fluid removal with haemodialysis can lead to cardiovascular collapse. Peritoneal dialysis, is used if heparin-free haemodialysis cannot be performed. It can compromise respiratory function because of intraperitioneal fluid accumulation. Large, non-resolving symptomatic effusions should be treated with instillation of intrapericardial corticosteroids (triamcinolone) after pericardiocentesis or subxiphoid pericardiotomy.

Pericardiectomy is indicated only in refractory, severely symptomatic patients.

Post –cardiac injury syndrome

Post–cardiac injury syndrome develops within days to months after cardiac/pericardial injury. It acutely provokes a greater antiheart antibody response (antisarcolemmal and antifibrillary antibodies). These antibodies appear to be pathogenic or may act in the presence of a dormant or concurrent viral infection. Treatment of this syndrome is based on administration an NSAID or colchicines.

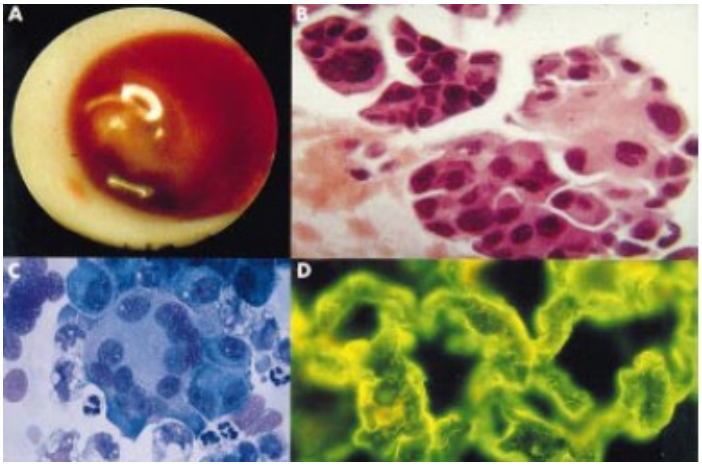
Pericarditis in myocardial infarction

Pericarditis may occur “early” (pericarditis epistenocardica) or be “delayed” (Dressler’s syndrome) after myocardial infarction. Epistenocardica pericarditis, caused by direct exudation, occurs in the first several days of almost half of transmural myocardial infarctions, although early thrombolytic treatment had decreased its incidence. Dressler’s syndrome occurs from one week to several months after myocardial infarction (also in subendocardial form) with manifestation similar to the post-cardiac injury syndrome. Cardiac tamponade may occur relatively early. Late constriction is rare but not surprising because of intrapericardial organization of exudates and blood .This also can produce loculated effusions. Ibuprofen is the agent of choice. Aspirin, up to 650mg every four hours for 2-5 days, corticosteroid treatment is used for refractory symptoms but can delay infarction healing.

Neoplastic pericarditis

It is 40 times more often caused by secondary than primary malignancies, most frequently by lung cancer, breast cancer, malignant melanoma, lymphomas, and leukemias. The diagnosis is based on pericardial fluid cytology and pericardial/epicardial biopsy findings. CT/MRI can reveal localization of the primary tumor or the metastases. Pericardial fluid

cytology is positive in 75-87% and pericardial biopsy in 27-65% of patients with malignant pericardial disease. However pericardial biopsies guided by pericardioscopy have a diagnostic value of 93.3 -97%. Increased concentrations of specific tumor markers (for example, carcinoembryonic antigen (CEA), α-feto protein (AFP), carbohydrate antigens CA 125, CA 72-4, CA15-3, CA 19-9, CD-30, and CD-25) may also suggest the diagnosis. In 60% of the patients with documented malignancy, pericardial effusion is caused by non-malignant diseases-for example radiation pericarditis or opportunistic infections.



Neoplastic pericarditis in Hodgkin's disease.

A. Pericardioscopy findings.
B. Epicardial Histology
C. Cytology of pericardial fluid
D. Immunofluorence of Epicardium

Systemic antineoplastic treatment is the baseline therapy and pericardiocentesis is required to relieve symptoms and establish diagnosis. Intra-pericardial instillation of cytostatic/ sclerosing agents is also useful in recurrent pericarditis. Recurrences are observed in 40-70% of patients with large malignant pericardial effusion. It is treated intrapericardial instillation of sclerosing agents, cytotoxic drugs, immunomodulators, systemic antitumour therapy, percutaneous balloon pericardiotomy, radiation therapy or surgical methods. Intrapericardial administration of cisplatin is effective in 83-93% of cases. It has the following side effects like fever (19%), chest pain (20%), and atrial arrhythmias (10%).Classic sclerotherapy after

intrapericardial instillation of tetracycline, doxycycline, minocycline, and bleomycin is an effective procedure. The development of constrictive pericarditis secondary to fibrosis remains a severe problem in long term survivors. Antineoplastic drugs (5-fluorouracil and aclarubicine) or immunomodulators (interferon, interleukin-2, OK-432) have been tested only in very small numbers of patients. Radiation therapy is very effective (93%) in controlling malignant pericardial effusion in patients with radiosensitive tumors such as lymphomas and leukemias. A percutaneous balloon pericardial window creates a pleuropericardial direct communication, which allows fluid to be drained into the pleural space. Pericardiectomy is rarely indicated, and mainly used for treating pericardial constriction or complication or previous procedures

Chylopericardium

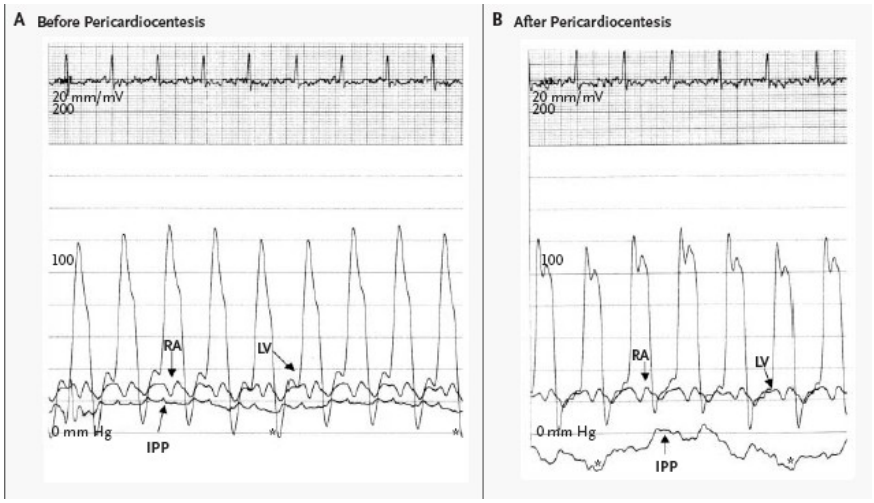
Chylopericardium refers to a communication between the pericardial sac and the thoracic duct caused by congenital anomalies, trauma, mediastinal lymphangiomas, lymphangiomatous hamartomas, mediastinal or pericardial lymphangiectasis, obstruction or anomalies of the thoracic duct, or caused iatrogenically during surgery. The chylous nature of the fluid is confirmed by its alkaline reaction, specific gravity between 1010 and 1021, Sudan III stain for fat, and the high concentrations of triglycerides (5-50g/L) and protein (22-60g/L).Enhanced CT alone or combined with lymphography, is a reliable diagnostic tool for identifying not only the location of the thoracic duct but also its lymphatic connection to the pericardium. Pericardiocentesis and diet are the principal therapy. After initial drainage the disorder may be resolved through dietary management with medium chain triglycerides alone. If further production of chylous effusion continues, surgical treatment is mandatory. Pericardio-peritoneal shunting by means of a pericardial window is a reasonable option. When the course of the thoracic duct had been precisely identified, ligation and resection of the thoracic duct just

above the diaphargam has proved to be the most effective treatment.

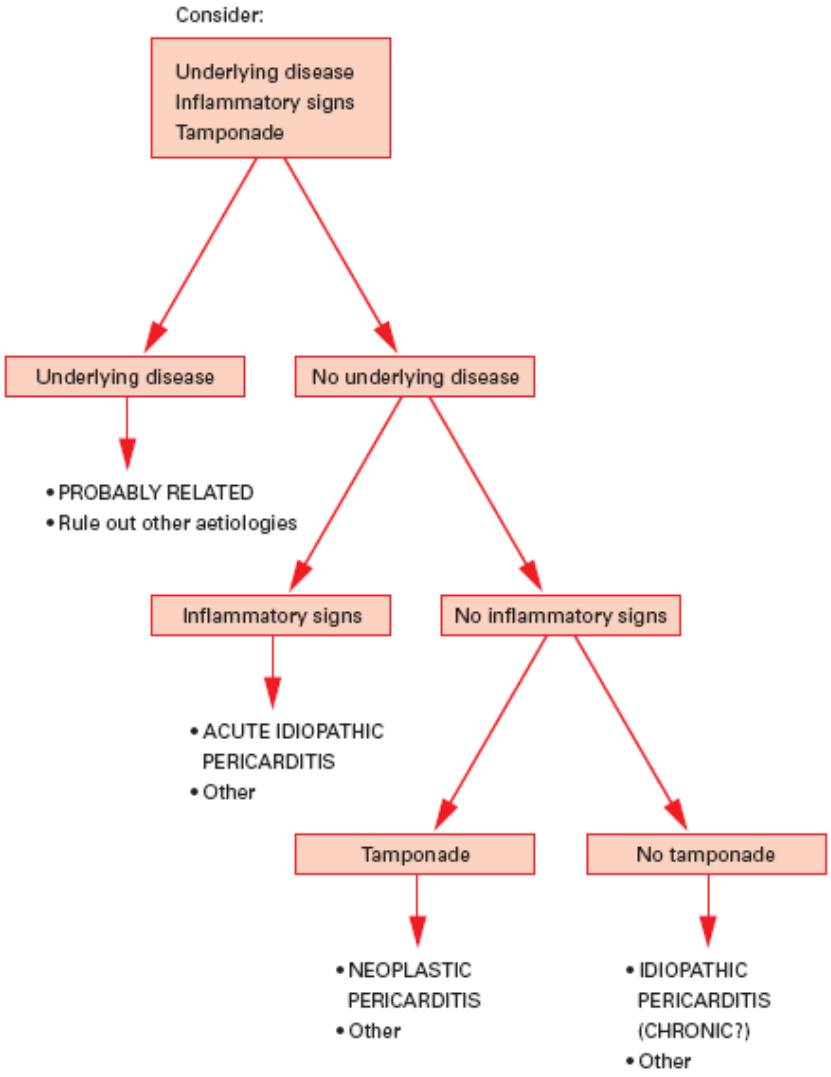
Idiopathic chronic pericardial effusion:

When a large pericardial effusion persists for more than three months, the prognosis, even in asymptomatic patients, is less good. Sagrista-Sauleda etal, have reported that up to 29% of such patients may develop unexpected, overt cardiac tamponade. Echocardiography can be used to assess the severity of pericardial effusion based on the size of effusions: (1) small (echo-free space in diastole <10mm); (2) moderate (at least ≥ 10mm posteriorly); (3) large (≥20mm); or (4) very large (≥20mm with compression of the heart). Colombo etal, consider effusions greater than 10mm by M mode echocardiography as large, effusions if they were greater than 5mm as small effusion. In the series by Sagrista –Sauleda etal, moderate effusions were defined as an echo-free space of anterior plus posterior pericardial spaces of 10-20mmm during diastole, and severs effusions as a sum of echo-free spaces greater than 20mm.

The trigger of tamponade is unknown, but hypovolaemia, paroxysmal tachyarrhythmia, and intercurrent acute pericarditis may precipitate tamponade, accordingly, these events should be vigorously managed. Anti-inflammatory drugs and corticosteroids are unsuccessful. Pericardiocentesis is indicated in patients with overt clinical tamponade, in patients with suspicion of purulent pericarditis, and in patients with idiopathic chronic large pericardia effusion.



Approach to pericardial effusion:



Cardiac Tamponade:

It is usual in acute pyogenic, tuberculous, and malignant pericarditis. Acute haemopericardium may cause tamponade following cardiac rupture after myocardial infarction, in dissection of the aortic root or after cardiac surgery. Metastatic carcinoma, as well as primary tumors, may present with tamponade.

Characteristics of Cardiac Tamponade:

Clinical presentation	Raised systemic venous pressure hypotension, pulsus paradoxus ,tachycardia, dyspnea or tachypnea with clear lungs
Precipitating factors	Drugs (cyclosporine, anticoagulants, thrombolytics, etc),recent cardiac surgery, indwelling instrumentation, blunt chest trauma, malignancies, connective tissue disease, renal failure, septicemia
ECG	Can be normal or non-specifically changed (ST-T wave), electrical alternans (QRS, rarely T), bradycardia (end stage), electromechanical dissociation (agonal phase)
Chest x ray	Enlarged cardiac silhouette with clear lungs
Mmode/2D echocardiogram	Diastolic collapse of the anterior RV free wall , RA collapse, LA and rarely LV collapse, increased LV diastolic wall thickness - “pseudo hypertrophy”, VCI dilatation (no collapse in inspiration), “swinging heart”
Doppler	Tricuspid flow increases and mitral flow decreases during inspiration (reverse in expiration) Systolic and diastolic flows are reduced in systemic veins in expiration and reverse flow with contraction is increased
M mode colour Doppler	Large respiratory fluctuations in mitral /tricuspid flows

Cardiac catheterization	Confirmation of the diagnosis and quantification of the hemodynamic compromise: RA pressure is raised (preserved systolic X descent and absent or diminished diastolic y descent) Intrapericardial pressure is also raised and virtually identical to RA pressure (both pressures fall in inspiration) RV mid-diastolic pressure raised and equal to the RA and pericardial pressures Pulmonary capillary diastolic pressure is slightly raised and may correspond to the RV pressure Pulmonary capillary wedge pressure is also raised and nearly equal to intrapericardial and right atrial pressure LV systolic and aortic pressure may be normal or reduced documenting that pericardial aspiration is followed by hemodynamic improvement detection of the coexisting hemodynamic abnormalities (LV failure, constriction, pulmonary hypertension) detection of associated cardiovascular diseases (cardiomyopathy)
RV/LV angiography	Atrial collapse and small hyperactive ventricular chambers
Coronary angiography	Coronary compression in diastole

Atypical Tamponade:

Low pressure tamponade occurs in the following conditions:

- ❖ Hypovolaemia
- ❖ Trauma
- ❖ Excessive diuretic

- Treatment
- Haemodialysis

Impending Cardiac Tamponade:

Some patients with pericardial effusion but without clinical tamponade show findings suggesting raised intra-pericardial pressure-namely, collapse of the right sided cardiac chambers. These findings indicate the “impending” cardiac tamponade.

Specifically, right atrial collapse had a low positive predictive value (50%) for clinical cardiac tamponade. However, these patients consistently show elevation of intrapericardial pressure when they undergo catheterization study. **Pericardiocentesis**

Pericardiocentesis is indicated for clinical tamponade, suspicion of purulent or neoplastic pericarditis, or for symptomatic patients, despite medical treatment for more than one week. Pericardiocentesis is indicated if additional diagnostic procedures are available (for example, pericardial fluid and tissue analyses, pericardioscopy, and epicardial/pericardial biopsy) to reveal the etiology of the disease and permit further treatment. Aortic dissection is a major contraindication; uncorrected coagulopathy, anticoagulant treatment, thrombocytopenia <50000/mm, and small, posterior, and loculated effusions are other relative contraindications for performing the procedure.

Pericardiocentesis may allow the resolution of effusion in a third of patients. When large effusion reappears after two pericardiocentesis, wide anterior Pericardiectomy has to be considered. The indications for surgical drainage are tamponade, either unresolved or relapsing after pericardiocentesis, and persistent active illness three weeks after hospital admission. Even the presence of echocardiographic right chamber collapse (suggesting raised intrapericardial pressure) does not warrant by itself pericardial drainage as most of these patients do not evolve

to overt tamponade. Routine pericardial drainage is not justified in the initial management of patients with large pericardial effusion without clinical tamponade, especially if the etiology is known. The exceptions would be those patients with suspected purulent or tuberculous pericarditis

Asbestos induced pericardial effusion and constrictive pericarditis:

Asbestos produces progressive fibrosis of the pericardium that is similar to diffuse pleural thickening and may be fatal. Both conditions may develop after relatively short or light exposure. In a study by Davies D etal, three cases have been reported. He reported a man with bilateral pleural thickening and plaques developed acute pericarditis and an effusion and he was treated by pericardiectomy. Two men died from constrictive pericarditis associated with bilateral pleural effusions and diffusion pleural thickening

Constrictive pericarditis:

Constrictive pericarditis can be defined as a syndrome (or syndromes) resulting from compression of the heart caused by rigid, thickened, and frequently fused pericardial membranes.

Patterns of Constrictive pericarditis:

Typical forms

Chronic (calcific, rigid shell)

Subacute (non-calcific, elastic)

Effusive-constrictive

Localized

Transient cardiac constriction

Occult constrictive pericarditis

Etiology

Prevalence of idiopathic CCP has varied form 24% to 61% in Indian studies. Tuberculosis remains a common cause of CCP in developing countries, with a reported incidence of 38%-83%. The etiology of CCP in the western world has now undergone a significant change, with tuberculosis reported in only 0%-1% of cases in some recent series. The leading identifiable causes of CCP are following cardiac surgery and radiation therapy, besides viral pericarditis. The incidence of CCP complicating cardiac surgery is between 0.025% and 0.15% of patients undergoing open heart surgery.

Pathogenesis

CCP may result from the progression of an acute pericarditis from a dry stage through an effusive, absorptive, a constrictive phase sequentially; or it may result from a smoldering fibrosis with no previous history of an acute pericarditis. It appears that tubercular pericarditis is a hypersensitivity reaction to antigens such as tuberculoproteins. The increased production of interferon gamma, tumor necrosis factor-alpha, and interleukin-1 and interleukin 2 in tubercular pericardial effusion suggest that the inflammation is orchestrated by T-helper -1 lymphocytes. T-lymphocytes and activated macrophages seem to play an important role in granuloma formation and fibrosis.

Clinical features:

Constriction is usually generalized but rarely may be localized, owing to constricting bands in the left or right atrioventricular grooves or round the entry of the superior or inferior vena cava. Most often it is due to tuberculous origin; a few are associated with rheumatoid arthritis, collagen vascular disease or uremia, or rarely following cardiac surgery or irradiation.

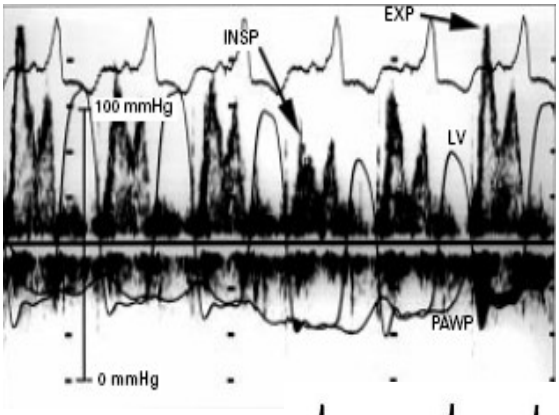
The clinical picture includes chronic fatigue and dyspnea, neck vein distension with a brisk diastolic collapse (“y”) of the jugular venous pulse, pericardial knock, enlarged liver, ascites, peripheral odema, and pleural effusion. Arterial pulsus paradoxus is usually absent. Atrial fibrillation is present in half of the patients. Constrictive pericarditis should be suspected in all patients with findings suggestive of right heart failure or ascites; diagnosis should be based on a triad of: a suggestive clinical syndrome; demonstration of a physiology of constriction/restriction; and demonstration of a thickened pericardium.

Characteristic of constrictive pericarditis:

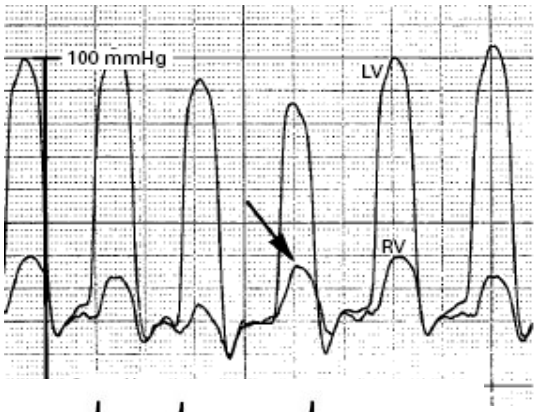
Clinical presentation	Severe chronic systemic venous congestion associated with low cardiac output, including jugular venous distension, hypertension with a low pulse pressure, abdominal distension, edema, and muscle wasting
ECG	Can be normal, or reveal low QRS voltage, generalized T wave inversion/flattening, LA abnormalities, atrial fibrillation, atrioventricular block, intraventricular conduction defects, or rarely pseudo infarction pattern
Chest x ray	Pericardial calcifications, pleural effusions
M mode/2D echocardiogram	Pericardial thickening and calcifications as well as the indirect signs of constriction: RA and LA enlargement with normal appearance of the ventricles, and normal systolic function Early pathological outward and inward movement of the interventricular septum Flattering waves at the LV posterior wall LV diameter is not increasing after the early rapid filling phase VCI and the hepatic veins are dilated with restricted respiratory fluctuations
Doppler	Restricted filling of both ventricles with respiratory variation >25% over the AV valves
TOE	Measurement of the pericardial thickness

MRI	Measurement of the pericardial thickness
Cardiac catheterization	"Dip and plateau" or "square route" sign in the pressure curve of the right and /or left ventricle Equalization of pressures in the range of 5mm Hg or less
RV/LV angiography	The reduction of RV and LV size and increase of RA and LA size During diastole a rapid early filling with stop of further enlargement ("dip-plateau")
Coronary angiography	In all patients over 35 years and in patients with a history of mediastinal irradiation, regardless of the age.

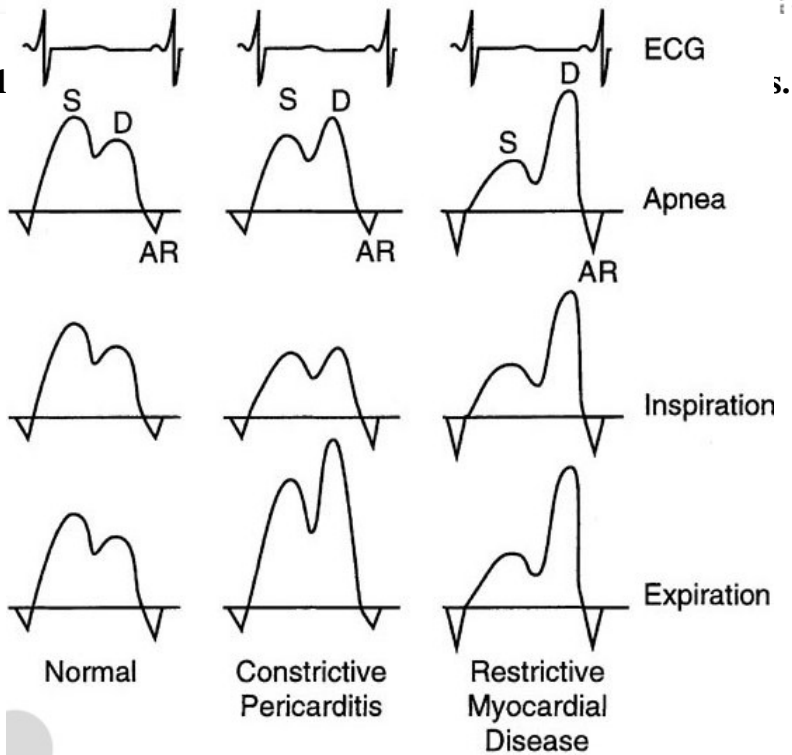
Mitral flow velocity Doppler

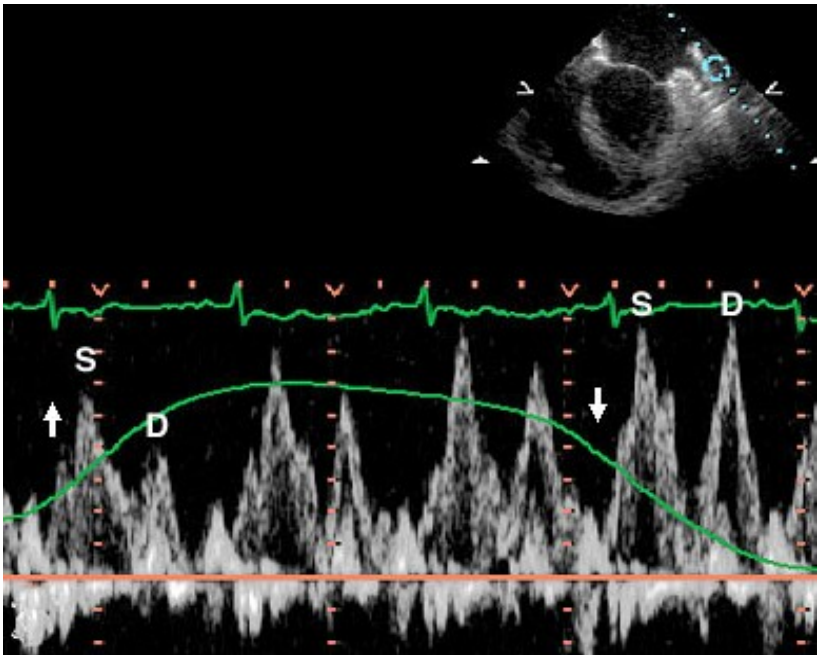


Cardiac Catheterisation



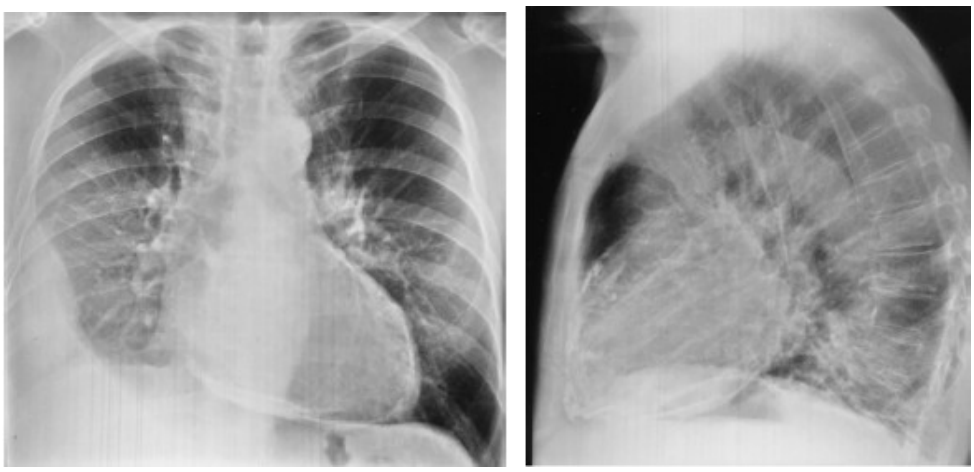
Pulsed wave Doppl **s.**





Pulsed wave Doppler of the pulmonary veins of a patient with constrictive pericarditis. Note the inspiratory decrease of the S and P waves and the expiratory increase due to interventricular dependence. (Inspiration is denoted by the upward arrow and expiration by the downward arrow).

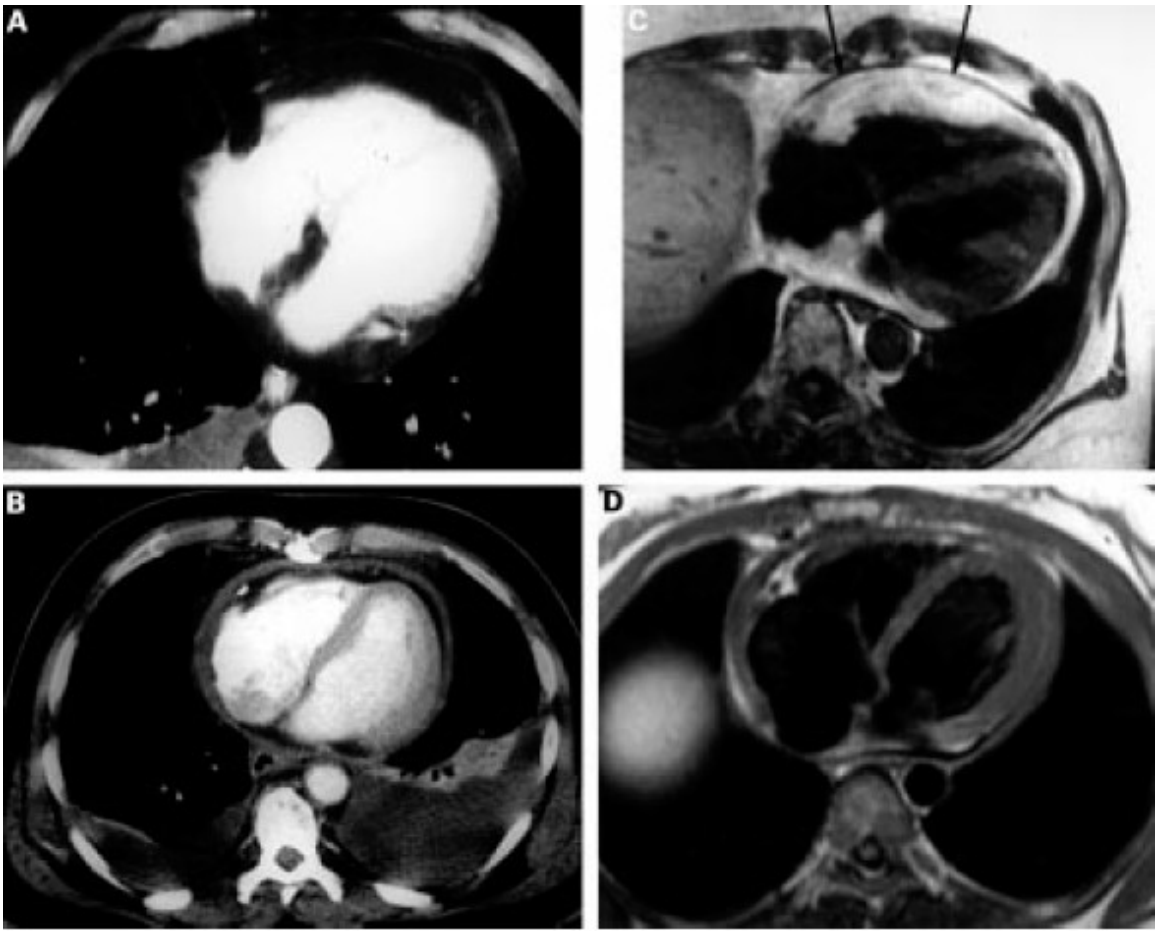
Pericardial calcification



Calcification occurred most frequently in patients with idiopathic constriction and patients longer duration of symptoms. The association of pericardial calcification with larger echocardiographic atrial dimension and volume is a new observation. The presence of calcification independently predicted perioperative death.

Cardiac MR:

Pericardial thickening of more than 3mm is usually taken as abnormal; the usual thickening of the pericardium being 1-2mm. Pericardial thickening on MRI was found to be 88% sensitive and 100% specific for detecting CCP. Additional findings suggestive of CCP, such as dilated inferior vena cava (IVC), enlarged atria, tubular-shaped ventricles, ascites and pleural effusion on MRI images. Myocardial tagging has also been suggested as a new method of mitral/tricuspid annular movement. As normal ventricles contract, there is a slippage between the myocardium and pericardium; however, once pericardial adhesions develop, this slippage is absent and the tag lines passing through the myocardium and pericardium are not deformed during the cardiac cycle. The presence of abnormal diastolic motion of the septum on cine MRI may also be a useful finding to diagnose CCP, and to distinguish it from RCM.



CCP with normal pericardial thickness was reported in 18% of 143 patients in a recently published series from the Mayo Clinic. These patients most commonly had post-surgical and post-radiation CCP. Their clinical characteristics were similar to patients with CCP with a thickened pericardium, and pericardiectomy was very effective in relieving the symptoms. Microscopy of the excised pericardium was abnormal in all the patients, which included the presence of focal fibrosis, focal calcification or inflammation

Comparison of subacute (elastic) and chronic (rigid shell) constrictive pericarditis

Subacute (elastic)	Chronic (rigid shell)
Paradoxical pulse usually present, other signs of interdependence usually prominent	Paradoxical pulse usually minimal or absent, other signs of interdependence less prominent
Usually an XY waveform ("M" or "W" waveform)	Y is predominant, X sometimes minimal
Dip-plateau pattern less conspicuous, because early diastolic nadir may not approach zero	Dip-plateau usually conspicuous, because early diastolic nadir often reaches zero.
Calcification usually absent	Calcification often present
Pericardial effusion sometimes present, generalized or loculated, constriction is by the visceral pericardium	Pericardial effusion absent. The two layers of pericardium are fused, and jointly constrict the heart.
P waves usually normal	P waves often wide, notched and low in amplitude
Atrial fibrillation or flutter rare	Atrial fibrillation or flutter common

Effusive –constrictive pericarditis:

Effusive- constrictive pericarditis is a clinical –hemodynamic syndrome in which there is constriction of the heart by the visceral pericardium in the presence of effusion in free pericardial space. This type of constrictive pericarditis was first characterized from the hemodynamic point of view by Hancock in his report on 13 patients submitted to Pericardiectomy. The hallmark of effusive-constrictive pericarditis is the demonstration of persistently raised right atrial and end diastolic ventricular pressure after the intrapericardial pressure is reduced to normal levels by removal of pericardial fluid. The etiologic spectrum includes idiopathic, following cardiac surgery, neoplasia and tuberculosis. Patients with effusive-constrictive pericarditis usually show a sub acute evolving clinical course with

inflammatory symptoms and signs. Most cases of effusive –constrictive pericarditis require Pericardiectomy. Effusive-constrictive pericarditis may be transient and may resolve spontaneously, especially in idiopathic cases.

Transient cardiac constriction:

Reported on 16 patients from a series of 177 with effusive acute idiopathic pericarditis in which features of constriction were detected in the phase of resolution of pericarditis. Clinical (pericardial knock on auscultation, “y” collapse in the jugular venous pulse) and echocardiographic (early diastolic septal notch) features of constriction were frequently present, and two patients had overt signs of venous congestion. In five patients, cardiac catheterization was carried out and disclosed features of constrictive pericarditis in all, either in the baseline state or after fluid challenge. After a mean period of 2.7 months the clinical and hemodynamic features of constriction spontaneously subsided. Although most patients with acute pericarditis proceed directly to complete resolution, in some recovery may be preceded by transient constriction; in some patients it may develop in to sub acute effusive-constrictive pericarditis, sometimes it may be followed by classical contractive pericarditis, or the condition may directly progress to sub acute or chronic constriction. Transient constriction would represent an intermediate link between uncomplicated recovery and the severe, irreversible types of constriction. Such an event occurred in 15% of patients with tubercular pericarditis in one report from South Korea. The resolution occurred within 2 months in the majority of patients. Perhaps transient CCP could occur even more often with purulent pericarditis.

Occult constrictive pericarditis:

The term occult constrictive pericarditis was introduced in 1977 by Bush and associates. They reported on 19 patients, most with idiopathic pericarditis, in whom physical and hemodynamic features of constriction were not apparent in their baseline state, but which were brought about by the rapid infusion of saline.

Patterns of Constrictive pericarditis in various pericardial diseases:

Patients with acute pericardial disease, severe sub acute constriction requiring pericardiectomy developed in 56% (9/16) and 35% (6/17) of patients with tuberculous and purulent pericarditis, respectively, and in 17%(2/12) of patients with neoplastic pericarditis. Only 2 of 177 patients with acute idiopathic pericarditis developed effusive-constrictive pericarditis requiring Pericardiectomy. Constriction was subacute, developing within the first six months of follow up. A patient with subacute constriction after acute pericardial disease is much more likely to have a specific pericarditis than an idiopathic pericarditis.

Transient constriction is much more frequent in patients with idiopathic pericarditis (with a prevalence of 20% when specifically sought) than in tuberculous or purulent pericarditis.

Pericardiectomy

Pericardiectomy is the only therapeutic approach that may remove permanent constriction, by aiming to resect the diseased pericardium as far as possible. The indications for surgery should be based upon clinical symptoms, echocardiography findings, CT/MRI, and heart catheterization. There are two standard approaches: (1) antero-lateral thoracotomy (fifth intercostal space); and (2) median sternotomy (faster access to the aorta and right atrium if

extracorporeal circulation becomes necessary. If severe calcified adhesions between the peri- and epicardium are present, or if the epicardium is generally affected (“outer porcelain heart”), surgery carries a high risk of either incomplete success or sever myocardial damage. An alternative approach in such cases may be “laser shaving” using an Excimer laser. Areas of strong calcification or dense scaring may be left as islands, separated from the other areas, to avoid bleeding. Major complications include acute perioperative cardiac insufficiency (regional bulging or complete dilatation of one or both ventricles) and ventricular wall rupture. The surgical mortality of pericardiectomy continues to be high, and has been generally reported to be 6%-12%. A subgroup of patients with calcific CCP had a mortality of 19% in a recent series. The negative predictors of survival after pericardiectomy include NYHA class IV, low-voltage ECG complex, markedly increased atrial pressure, associated organ failure, and post-radiation CCP. The occurrence of low cardiac output following CCP is often a reflection of the chronicity of CCP and associated myocardial atrophy. Acute cardiac dilatation and failure following pericardiectomy may occur unpredictably. Preoperative inotropes for 48 hours, and preoperative digitalization are often used to reduce the chances of postoperative heart failure but their effectiveness is not clear. Multiple incisions into the fibrous epicardium while protecting the myocardium and coronary arteries are useful in relieving the constriction (waffle procedure). In patients with extensive calcific plaques, where large plaques do not permit the development of cleavage planes, wedge incisions that reach up to the epicardium help release constriction. More recently, ultrasonic decalcification has been used in tough calcific lesions. Complete normalization of cardiac hemodynamic after the procedure has been reported in only 60% of patients. Postoperative low cardiac output should be treated by fluid substitution and catecholamine, high doses of digitalis, and, in the most severe cases, intra –aortic balloon

pump.

Relapsing pericarditis:

The term relapsing pericarditis includes two clinical types, the incessant type and the intermittent type of recurrent pericarditis. The term incessant applies to those patients in which discontinuation or attempts to wean from anti-inflammatory treatment nearly always ensure a relapse in a period of less than six weeks. This situation can be seen after discontinuation of the usual anti-inflammatory drugs (aspirin, indomethacin, or ibuprofen).The term “intermittent” refers to those patients with symptom-free intervals conventionally longer than six weeks without treatment. Their frequency in clinical series is between 8-80% with the average of 24%

The most typical from or relapsing pericarditis occurs after a first episode of idiopathic benign pericarditis, presumably of viral origin. The viruses most frequently implicated are Enterovirus, mainly Coxsackie’s B. Autoimmune response can certainly play a role in the pathogenesis of recurrent idiopathic or post viral pericarditis. Relapsing pericarditis can also occur in the post-myocardial and post-pericardial injury syndromes. Relapses after open heart surgery seem to more frequent in children and adolescents, especially after atrial open heart surgery seem to be more frequent in children and adolescents, especially after atrial septal defect closure. Genetic factors may play a role in the development of a relapse as well. An immunopathologic mechanism seems to be evident in the relapsing pericarditis of vasculitis or connective tissue disease, especially disseminated lupus erythematosus. Neoplastic pericarditis can shown an oscillating clinical course, and, occasionally, apparently self limited pericarditis with subsequent reappearance of pericardial manifestations.

The pattern of relapsing pericarditis is quite characteristic in that the index attack usually is the most severe, while subsequent episodes are milder. So in some patients the clinical

manifestations in relapses are limited to “pericardial” pain only. In the historical series by Fowler and Harbin that included 31 patients with recurrent acute pericarditis, all patients had pericardial friction rub and evidence of pericardial effusion or characteristic electrocardiographic changes in the index attack, while these manifestations were entirely absent in subsequent relapses in seven patients. In these patients the evidence of pericarditis (apart from pericardial pain) was provided by an increased erythrocyte sedimentation rate, while blood cell counts, or fever. In the series by Fowler and Harbin tamponade was present in the initial attack in three patients, but in none of the recurrences. The drug of first choice is aspirin, 250-500mg orally every 4-6hours. Paracetamol (acetaminophen), 500-750mg every 4-6hours, can be successfully used in patients with mild to moderate symptoms. In patients with severe symptoms, the most effective drugs are indomethacin (25-50 mg every 6-8hours) and ibuprofen (800mg every 6-8hours). Corticosteroids are very effective drugs that quickly control pain and pericardial effusion. Corticosteroid administration may favour the relapses whenever the dose is diminished, leading to recurrent pericarditis. Prednisone can be quickly reduced from initial high doses (1mg/kg/day) to the threshold level below which symptoms will probably recur (usually 5-15mg/day). From this point on, the doses of prednisone should be reduced very slowly in decrements of as little as 1mg.Doses can usually be reduced at intervals of between one week and two months, and can be given on alternate day. Pericardiectomy should be considered to be the last resort in patients with severe relapsing pericarditis in whom an adequate drug treatment has failed.

AIM

To find out the etio-pathological and clinical profile of patients with Chronic Pericarditis.

OBJECTIVES

- To analyze the etiological profile patients with chronic pericarditis.
- To describe the pathological findings in various chronic pericarditis.
- To elicit the clinical profile of chronic pericarditis.

MATERIAL AND METHODS

Study Design:

This study is a descriptive study. This study was conducted to describe the various etiologies in patients with chronic pericarditis.

Total no. of patients:

There were totally 50 patients were included in this study.

Place of study:

This study was conducted in Government Rajaji Hospital, Madurai. 50 consecutive patients who were attending Cardiology out patients department fulfilling the inclusion criteria were included in this study.

Study Period:

This study was conducted from August 2003- August 2005.

Inclusion Criteria:

- Patients with clinical features of chronic pericarditis which lasted for more than three months were included.
- All patients with the diagnosis chronic pericardial effusion; Chronic Cardiac Tamponade; Chronic Constrictive pericarditis were included in this study.

Exclusion Criteria:

- All patients with clinical features of pericarditis which lasted for less than three months were excluded.

Consent:

We got informed consent in all our study population.

All patients were underwent a proper systematic clinical examination as mentioned in the Proforma immediately after the enrollment. Complete Hemogram (Hemoglobin; Total WBC Count; Differential Count; ESR) Biochemical investigations (Blood urea; Serum creatinine) were done in all patients. Chest X-ray was taken in both PA & Lateral views in all patients.

Surface ECG:

Surface ECG was taken in all patients. ECG was analyzed for the presence of low voltage complexes, atrial enlargement, arrhythmias. Low voltage complex was defined if QRS voltage in leads I, II, III each less than 0.5 mV, or V1 to V6 each less than 1.0 mV.

Echocardiographic examination:

In all patients 2D & M Mode & Doppler examination was done. In M Mode we analyzed the septal motion, septal notch; septal bounce; pericardial fluid quantification; pericardial thickness & posterior wall movement. The severity of pericardial effusion was classified based on the size of effusions: (1) small (echo-free space in diastole <10mm); (2) moderate (at least ≥ 10mm posteriorly); (3) large (≥20mm); or (4) very large (≥20mm with compression of the heart. In 2D Echo we specifically we looked for the presence of early diastolic Right atrial collapse and the presence of collapse of the free wall of Right ventricle. The Doppler examination was done to find out the flow variation during the both phases of respiration. We analyzed the respiratory flow variation in mitral valve; Tricuspid valve. We also analyzed the respiratory flow variation in hepatic vein and Pulmonary vein.

Transesophageal Echocardiographic examination was done in all patients. This modality

was used mainly to assess the respiratory flow variation in pulmonary vein and also for the accurate measurement of pericardial thickness. Pericardial biopsy was taken in 27 patients. In all 27 patients the biopsies were analyzed by histopathological examination. Bacterial; Tuberculous & fungal cultures were taken in all 27 patients. DNA PCR for mycobacterium Tuberculosis was done in 5 patients.

Polymerase chain reaction to detect Mycobacterium tuberculosis in Pericardial tissue (DNA-PCR):

Mycobacterium tuberculosis infection can be identified by detecting M.tuberculosis specific DNA sequence in tissue samples. **IS6110**, an insertion sequence (transposon) specific for M.tuberculosis and M.bovis and **devR** gene responsible for the synthesis of DevR, (a protein involved in the signal transduction system of Mycobacterium tuberculosis; which also found in Mycobacterium bovis) are the widely used genome sequences.

Methods:

Genomic DNA was extracted from the pericardial tissue using the Phenol/Chloroform/Isoamylalcohol method and quantitated. PCR was performed for the IS6110 insertion element and devR gene in a final volume of15 uL with 100 ng of DNA as template. The PCR product was electrophoresed in a 1.5 % agarose gel for 30 minutes at 100 volts. The Agarose gel was then visualized under UV light and documented using a Kodak digital image analysis system.

Table – 1: Baseline Characteristics

Total no. of patients	50
Male	25 (50%)
Age in years (mean ± SD)	34 ± 16
Mean duration of symptoms (in months)	4.2 ± 4.3
Clinical Profile	
Dyspnea class II to IV	44 (88%)
Facial puffiness	30 (60%)
Pedal edema	30 (60%)
Chest pain	25 (50%)
Fever	22 (44%)
Hypothyroidism	6 (12%)
Chronic renal failure	4 (8%)
Previous H/o tuberculosis	4 (8%)
H/o rheumatic fever	1 (2%)
Acute myocardial infarction	1 (2%)
H/o irradiation	Nil
Mean pulse rate (per min)	93 ± 16
Elevated jugular venous pressure	33 (66%)
Pulses paradoxus	13 (26%)
Pericardial knock	9 (18%)

Table 1 describes the baseline characteristics of the study population. Totally fifty patients were included in this study. Out of them 25 (50%) patients were male. The mean age of

our study population was 34 ± 16.3. Dyspnea was the predominant symptom in our study population. 44 (88%) patients had Dyspnea. Facial puffiness was present in 30 (60%) patients. Only 4 patients (8%) had previous history of Tuberculosis. 4 patients (8%) had evidence of chronic renal failure and 6 (12%) patients had evidence of hypothyroidism. 13 patients (26%) had pulsus paradoxus and 9 patients had pericardial knock.

Table – 2: Patterns of Chronic Pericarditis

Pericardial Disease	Total No. of Patients
Pericardial Effusion (PE) Small	2 (4%)
Moderate	11(22%)
Large	14 (28%)
Constrictive Pericarditis (CCP)	12(24%)
Cardiac Tamponade (CT)	11(22%)

Table 2 describes the profile of our study population. 12 (24%) had constrictive pericarditis and 11 (22%) had Cardiac Tamponade. 27 patients (54%) had pericardial effusion. Out of 27patients 14(52%) patients had large pericardial effusion; 11(41%) patients had moderate pericardial effusion and 2(7%) patients had small pericardial effusion.

Table – 3: Clinical Profile

	Pericardial Effusion (PE)	Constrictive Pericarditis (CCP)	Cardiac Tamponade (CT)
Age in Years (Mean ± SD)	39 ± 16	20 ± 8	34 ±12
Male (%)	9 (33%)	9 (75%)	7 (64%)
Duration of symptoms (months)	4.8 ± 5.6	4.1 ± 2.6	3.0 ± 0.2
Previous evidence of tuberculosis	1 (4%)	2 (16%)	1 (9%)
Dyspnea	25 (92%)	11 (91%)	8 (72%)
Facial Puffiness	16 (59%)	8 (66%)	6 (54%)
Pedal edema	16 (59%)	8 (66%)	6 (54%)
Chest pain	13 (48%)	6 (50%)	6 (54%)
Fever	7 (26%)	11 (91%)	4 (36%)
No.of Patients with Pulsus Paradoxus	4 (15%)	Nil	9 (82%)
Mean Jugular Venous Pressure (cm)	6.9 ±2.6	7.5 ±2.2	9.8 ± 1.3
No.of Patients with Pericardial Knock	Nil	9 (75%)	Nil

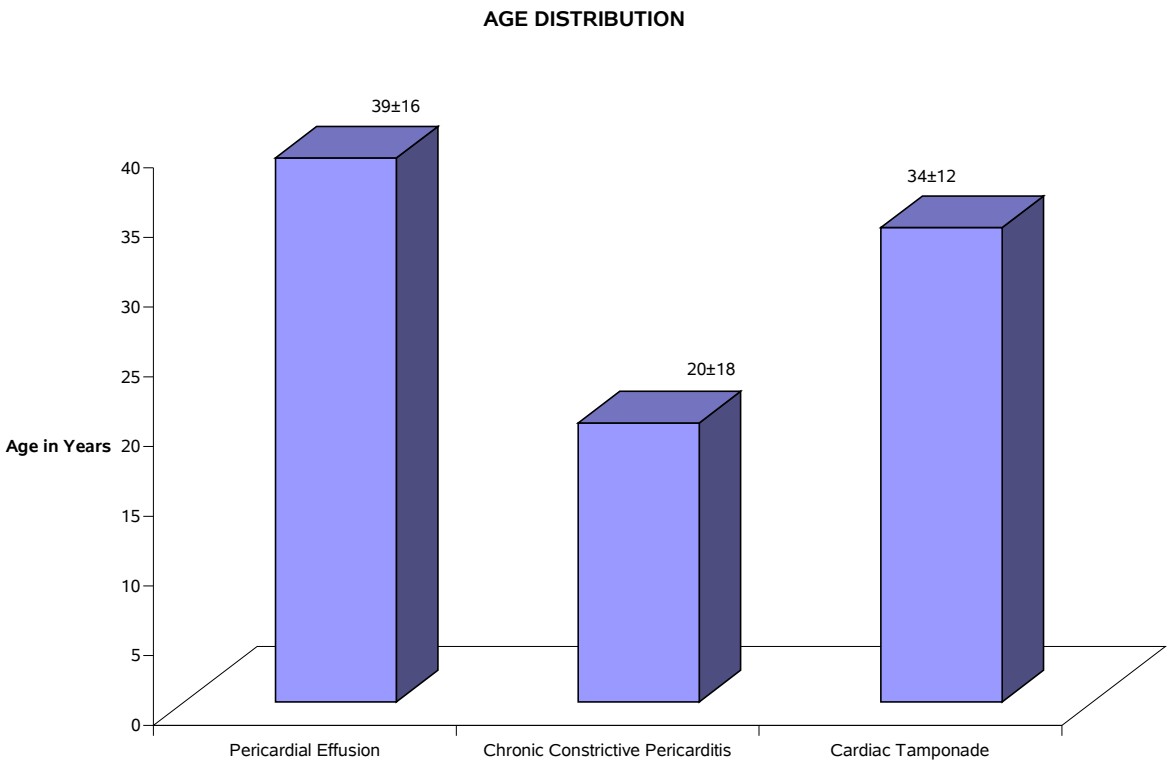
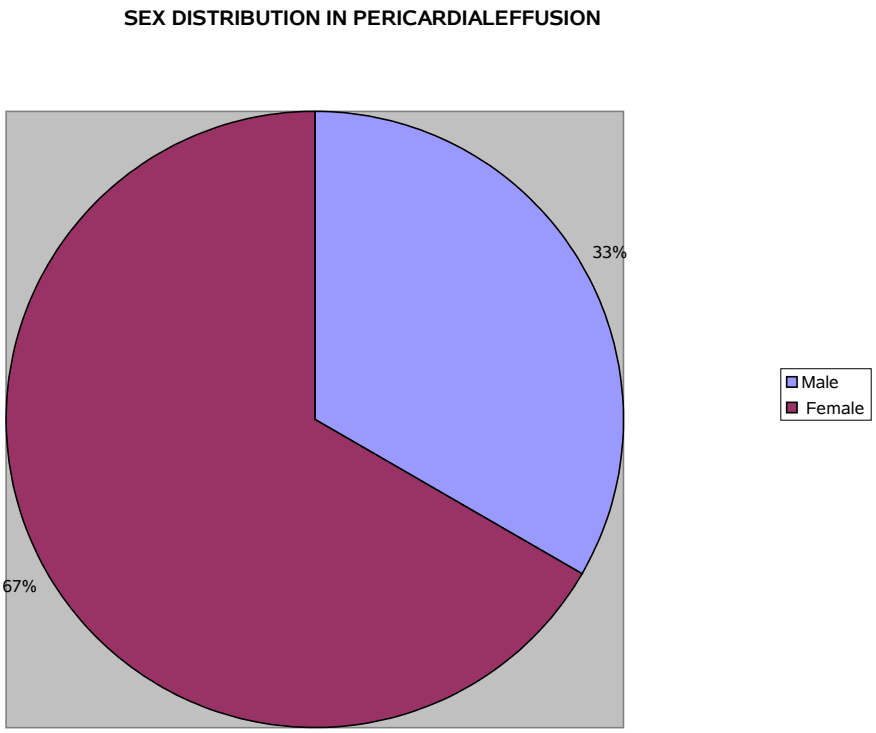
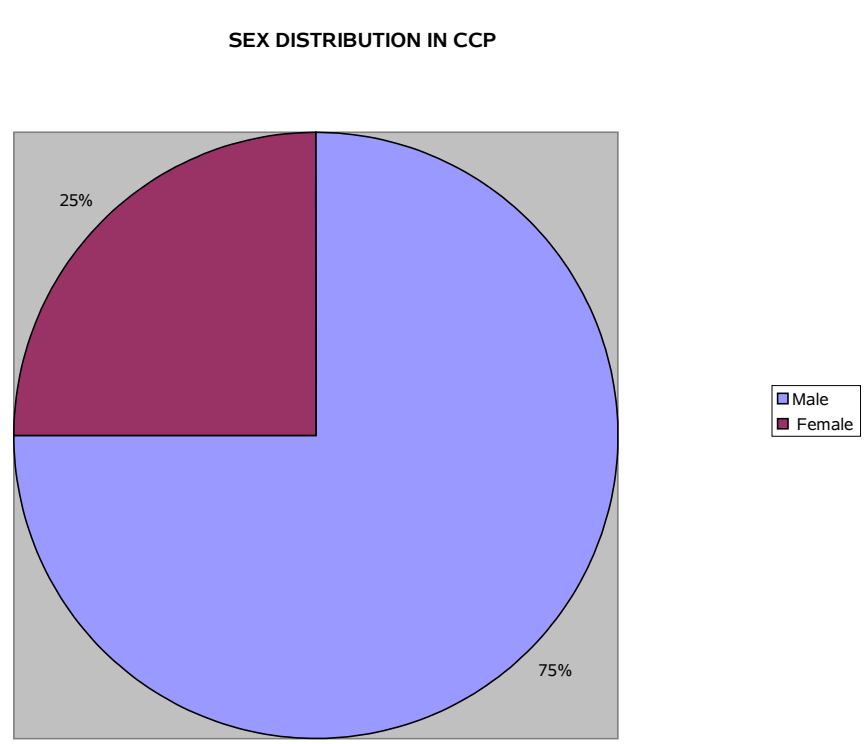


Fig: 1 Age distribution in the three patterns of chronic pericarditis

Fig 2 -4 the sex distribution in the three forms of chronic pericarditis.





SEX DISTRIBUTION IN CARDIACTAMPONADE

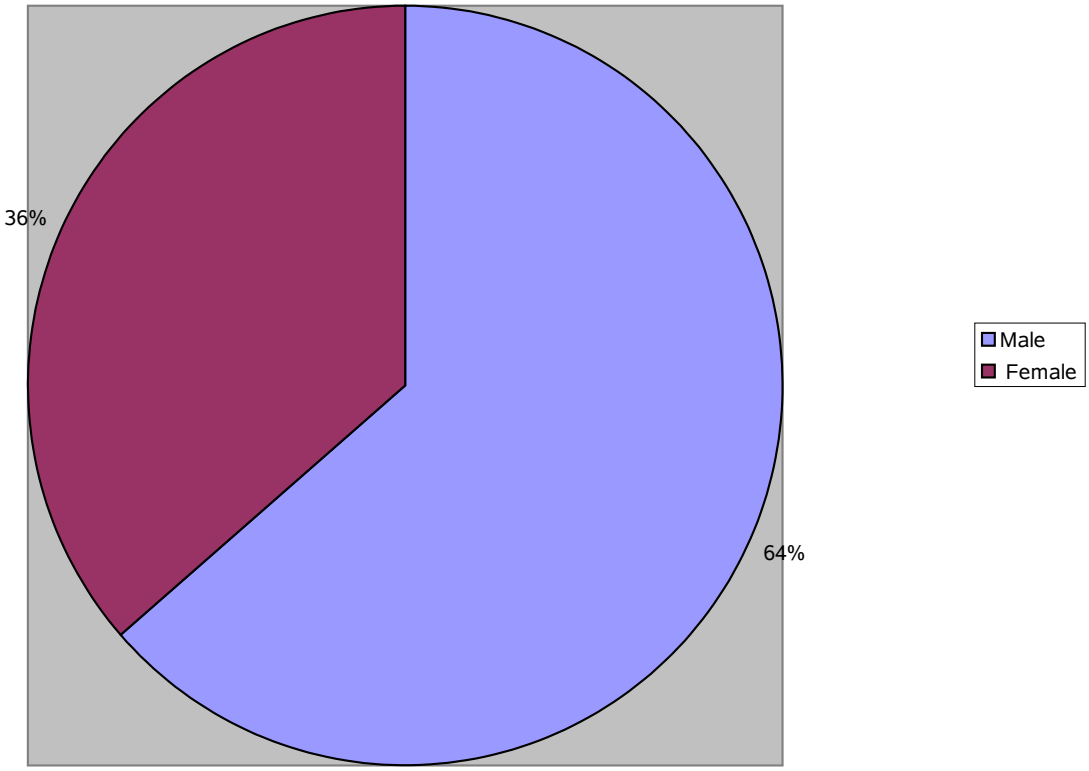


Table 3 showed the clinical profile of our patients. We divide our study population in to three groups' namely pericardial effusion; chronic constrictive pericarditis; Cardiac Tamponade. The mean age of patients in three groups was 39, 20, and 34 respectively (Fig 1). Patients in Constrictive pericarditis group were younger age group than the others. The predominant study population in constrictive pericarditis& cardiac Tamponade were male (Fig 2 & 3). In pericardial effusion group predominantly they were female (Fig 4). The mean duration of clinical presentation prior to the study was 4.8; 4.1; 3 months respectively. 16% of patients in the constrictive pericarditis group had previous evidence of Tuberculosis. 9 Patients (82%) in cardiac tamponade and 4(15%) in severe pericardial effusion group had pulsus paradoxus. The mean JVP was 6.9; 7.5; 9.8 respectively. 9 Patients (75%) in constrictive pericarditis group had pericardial knock.

Table – 4: Etiological Profile

Diseases	PE	CT	CCP
Tuberculosis	1(4%)	2(18%)	5(42%)
Rheumatic fever	1(4%)	NIL	NIL
Irradiation	NIL	NIL	NIL
Myocardial infarction	1(4%)	NIL	NIL
Chronic renal failure	4(15%)	NIL	NIL
Hypothyroidism	6(22%)	NIL	NIL
Idiopathic	14(51%)	9(82%)	7(58%)

(PE = Pericardial effusion; CT = Cardiac Tamponade; CCP = constrictive pericarditis)

Table 4 showed the etiological profile of study population (Fig 5). Most common

etiology in all the three groups was idiopathic. In CCP 7 patients (58%) belonged idiopathic cause and in 5 patients (42%) it was due to tuberculous infection (Fig: 6). In patients with cardiac Tamponade in 9 patients (82%) it was due to idiopathic cause and the rest of the 2 patients (18%) it was due to tuberculous infection (Fig 7). In patients with pericardial effusion 14 patients (51%) were idiopathic and 6 patients (22%) were hypothyroid and 4 patients (15%) were in chronic renal failure. The rest of 3 patients belonged to Myocardial Infarction; Rheumatic fever and tuberculous etiology. (Fig 8)

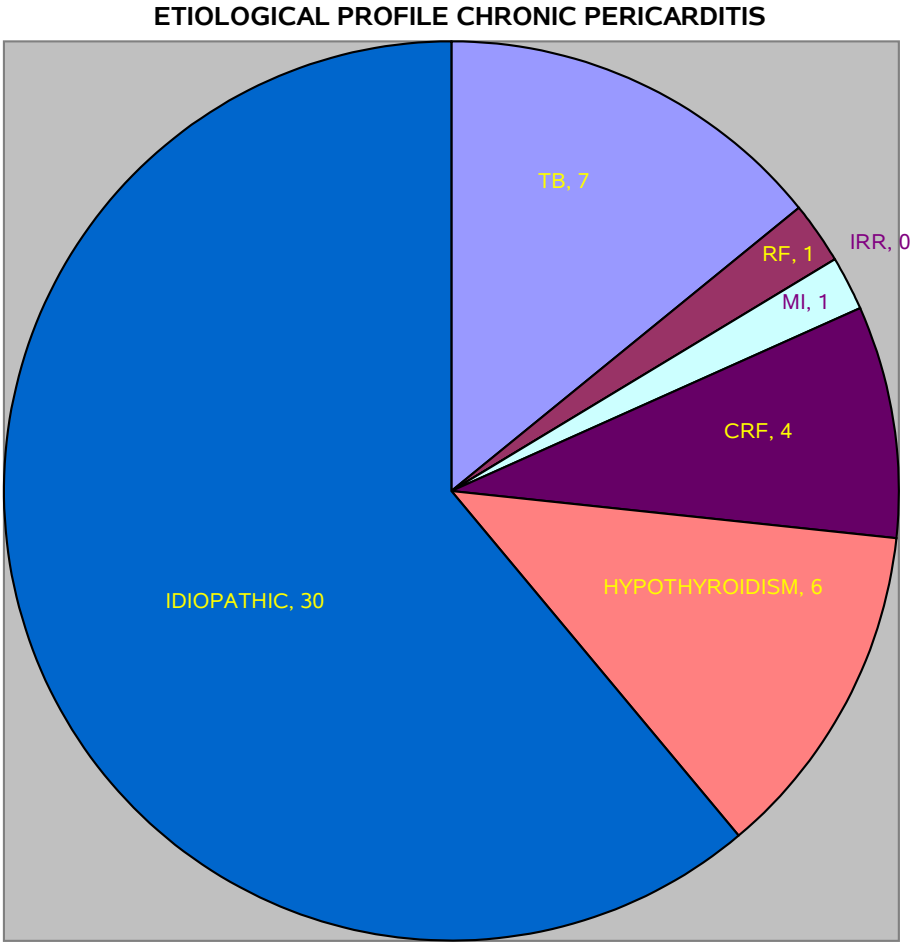


Fig: 5 the Etiological profile of chronic pericarditis

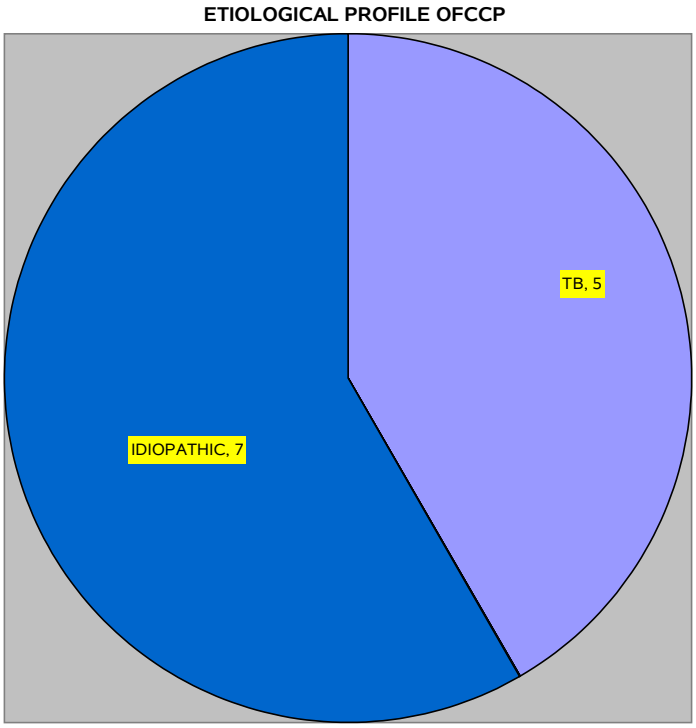


Fig: 6 the Etiological profile of constrictive pericarditis

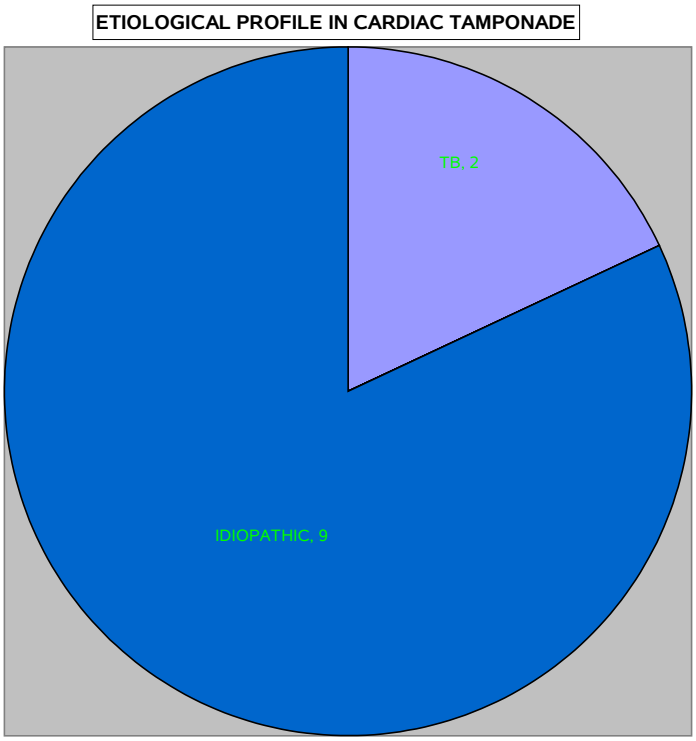


Fig: 7 the Etiological profile of cardiac Tamponade.

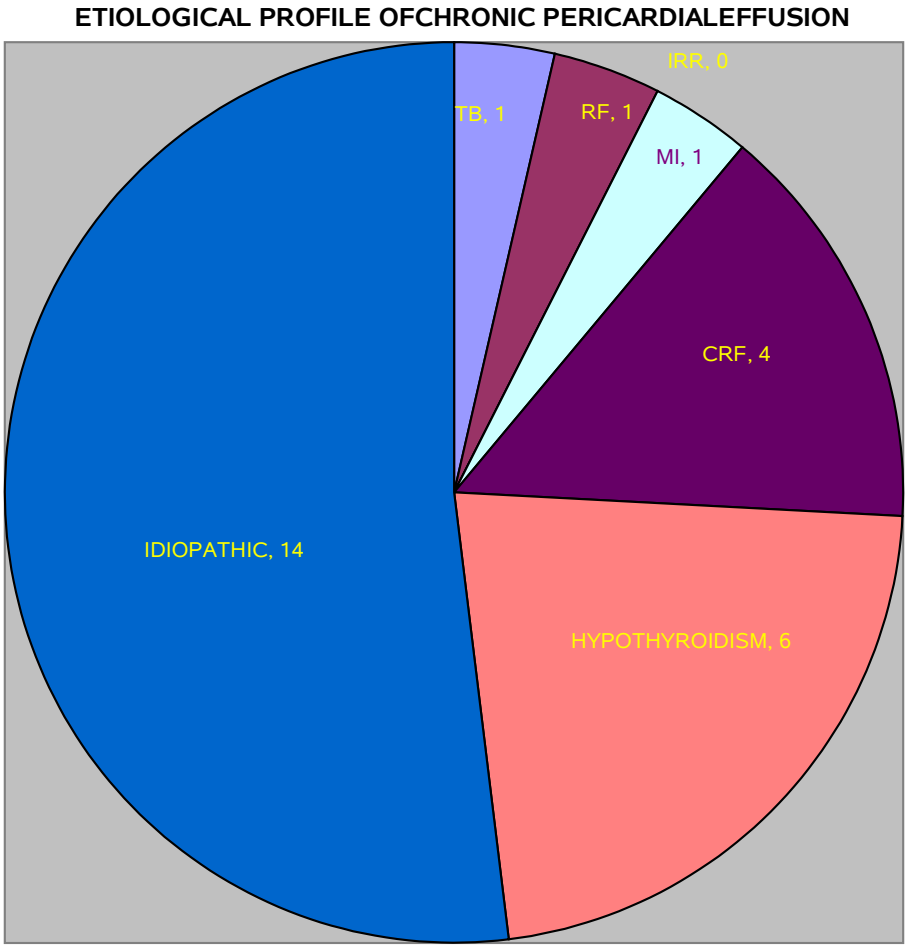


Fig: 8 The Etiological profile of chronic pericardial effusion

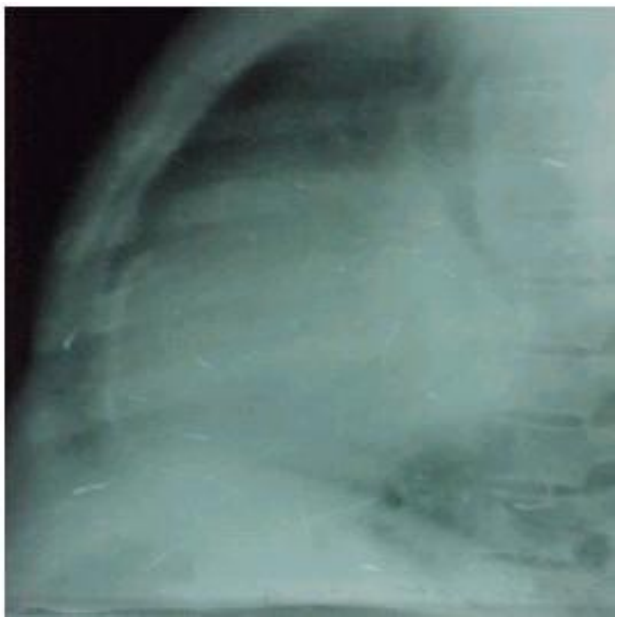


Fig: 9 Chest X-ray lateral view in a patient with CCP showing pericardial calcification

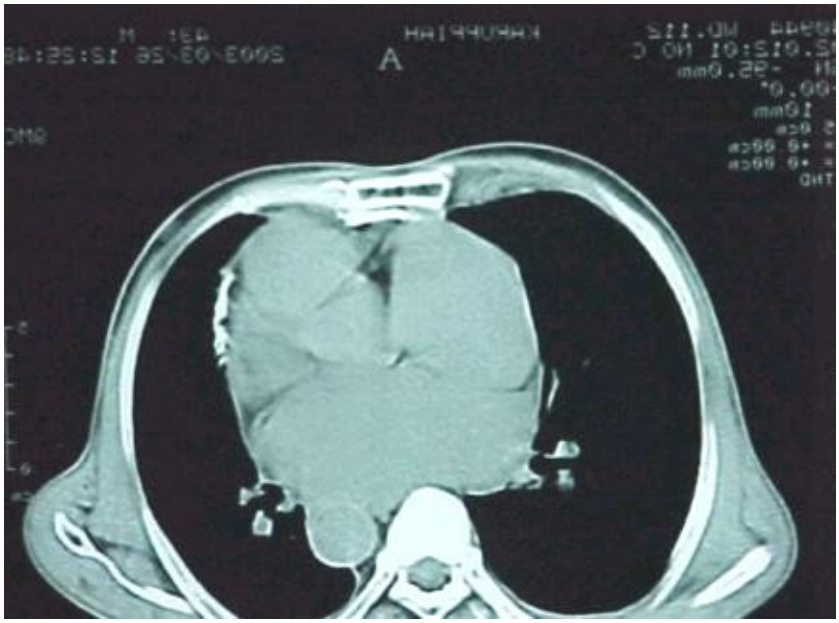


Fig: 10 CT scan showing pericardial calcification in a patient with CCP.

Table 5: Baseline Investigations

	PE	CCP	CT
ESR (mm/hour)	26 ± 21.1	35.6 ± 18	41.6 ± 24.6
CTR (%) in chest X-ray	60 ± 7.4	54.4 ± 3.3	62.3 ± 3.37
Pericardial calcification in X -ray	Nil	3(25%)	Nil
No. of patients with Low voltage complexes in ECG	15 (55%)	6(50%)	9 (82%)

(PE = Pericardial effusion; CT = Cardiac Tamponade; CCP = constrictive pericarditis; CTR = Cardiothoracic ratio.)

Table 5 showed the comparison of baseline investigations of our study population. ESR was high in patients with cardiac Tamponade. The mean CTR was also high in patients with cardiac Tamponade. 3 patients (25%) showed evidence of pericardial calcification (Fig 9&10). 9 Patients (82%) had low voltage complex in ECG in patients with cardiac Tamponade.

Fig: 11 Apical 4 chamber view showing diastolic Right Atrial Collapse in a patient with cardiac Tamponade.

Fig 12 Apical 4 chamber view showing large pericardial effusion

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Fig 13. Parasternal long axis view showing dilated left atrium with thickened pericardium

Fig 14. Parasternal short axis view showing thickened pericardium with mild pericardial effusion.

Fig: 15 Respiratory flow velocity variation in mitral valve in a patient with CCP.

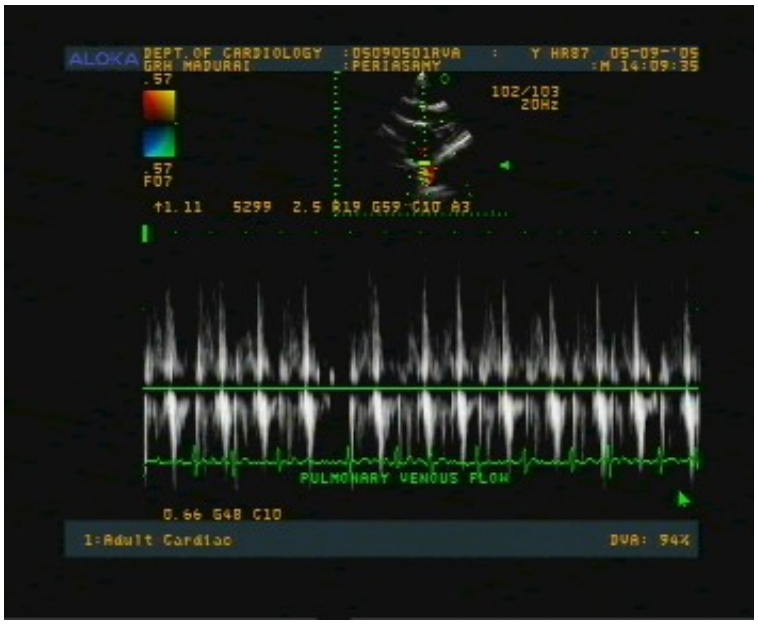


Fig: 16 Respiratory flow velocity variation in pulmonary venous flow in a patient with CCP.

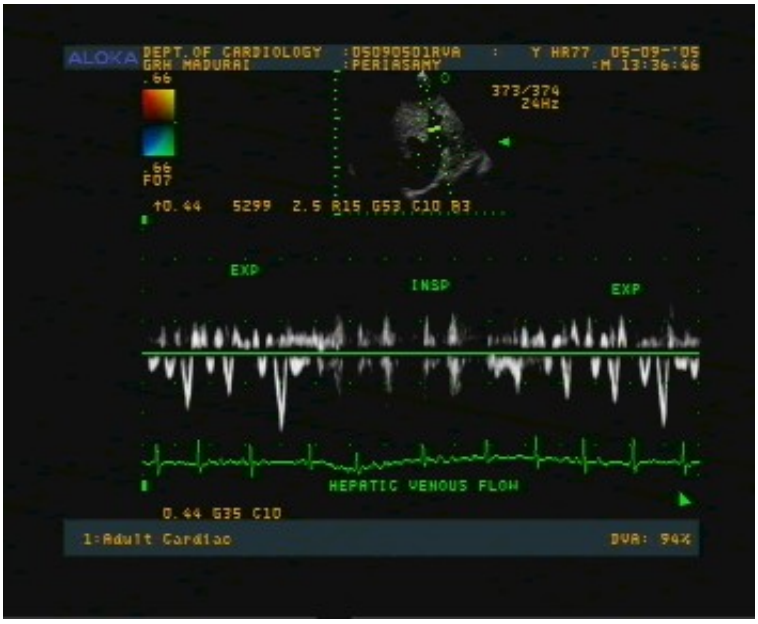


Fig: 17 Respiratory flow velocity variation in hepatic venous flow showing prominent diastolic flow reversal in a patient with CCP.

Table 6: ECHO characteristics of chronic Pericarditis

Variables	PE	CCP	CT
Right Atrial Collapse	NIL	Nil	11
Right Ventricular Collapse	NIL	Nil	11
Septal Bounce	NIL	12	Nil
Flat Posterior Wall	NIL	12	Nil
Pericardial Thickness (mm)	2.29 ± 0.4	4.4 ± 0.5	2.45 ± 0.5
Pericardial Effusion (mm)	18.26 ± 5	7.6 ± 2.9	25.4 ± 4.9
MV flow velocity respiratory variation (Mean %)	9.2 ± 2.3	28.3 ± 2.4	24.5 ± 3.6
Total no. of patients with prominent diastolic flow reversal in hepatic vein during Expiration.	15 (55%)	11 (91%)	11 (100%)

(PE = Pericardial effusion; CT = Cardiac Tamponade; CCP = constrictive pericarditis)

Table 6 showed Echocardiographic features of patients in chronic pericarditis (Fig 11-

17). The mean pericardial thicknesses in all the three groups were 2.29; 4.4 and 2.45mm. The mean pericardial thickness was high in patient s with constrictive pericarditis group. The mean MV flow velocity respiratory variation was high in patients with constrictive pericarditis.

Fig: 18 per operative findings in a patient with CCP

Table7: Peroperative Findings

	PE	CCP	CT
Mean amount of fluid (ml)	466 ± 115	283 ± 135	650 ± 92
No. of pts. with Hemorrhagic fluid	1 (33%)	1 (17%)	6 (54%)
Mean pericardial thickness (mm)	-	4.4 ± 0.5	2.4 ± 0.5

(PE = Pericardial effusion; CT = Cardiac Tamponade; CCP = constrictive pericarditis)

Table 7 showed peroperative findings in our study population. The mean amount of pericardial fluid was high in patients with cardiac Tamponade. The mean amount of pericardial fluid in patients with cardiac tamponade was 650 ± 92ml. 6 patients (54%) in cardiac tamponade group had hemorrhagic pericardial effusion. The mean pericardial thickness was high in patients with constrictive pericarditis. It was 4.4 ± 0.5mm in patients with constrictive pericarditis and 2.4 ± 0.5mm in patients with cardiac tamponade (Fig 18).

Table 8: DNA PCR for Mycobacterium Tuberculosis

	No. of patients	Positive
Constrictive Pericarditis (CCP)	5	4

DNA PCR for Mycobacterium Tuberculosis was done in 5 patients in the constrictive pericarditis group. Out of them 4 patients (80%) were found positive for tuberculous infection (Fig 19-20).

Fig 19: Electrophorogram of IS6110 specific PCR product using DNA from different pericardial samples with M.tb DNA as positive control

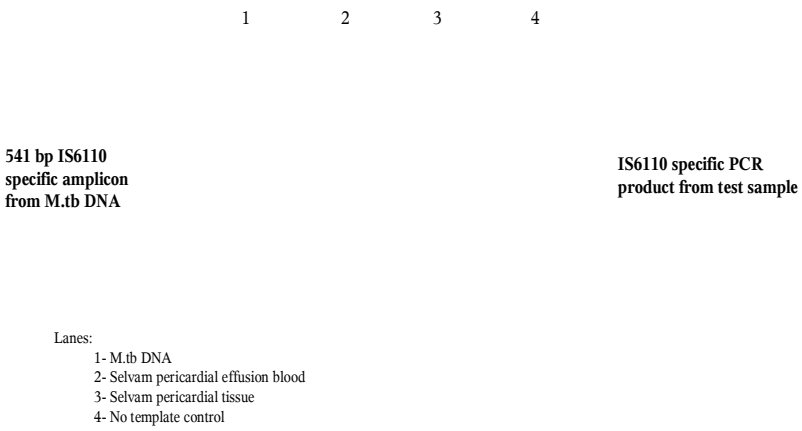
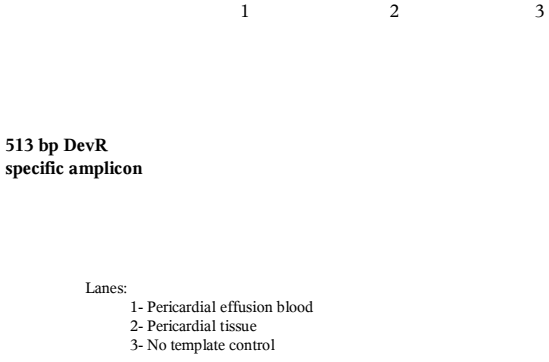


Fig 20: Electrophorogram of DevR specific PCR product using DNA from different samples of a single pericarditis patient



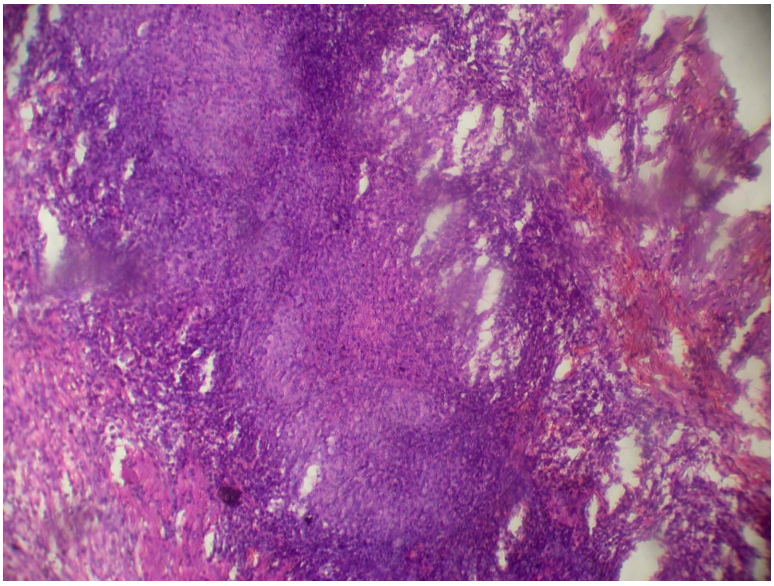


Fig: 21 histopathological examination of pericardium in a patient with CCP showing Caseating granuloma.

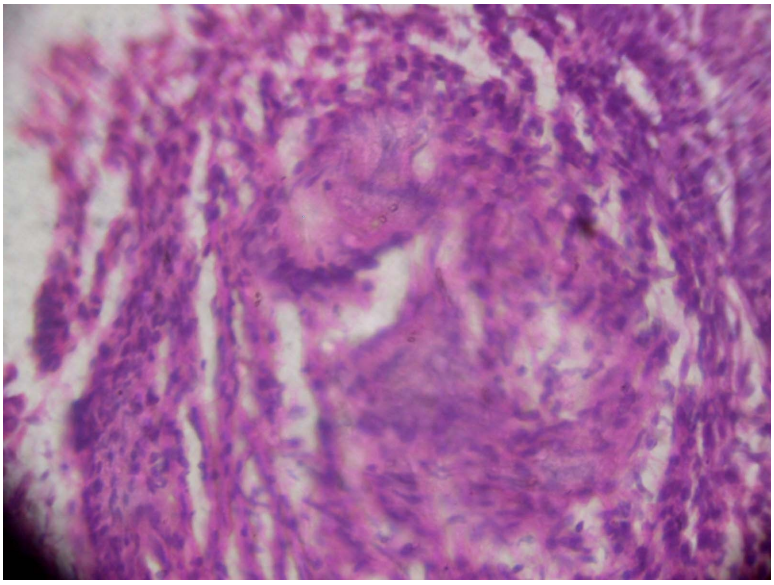


Fig: 22 histopathological examination of pericardium in a patient with CCP showing granuloma with Langhans giant cell

Table 9: Histopathological Examination

Biopsy	PE	CP	CT
NSPI	3 (100%)	7 (58%)	9 (82%)
Caseating Granuloma	Nil	5 (42%)	2 (18%)

(NSPI = Non Specific Inflammation)

Pericardial biopsy was taken in 3 patients in the large pericardial effusion group and all of them showed non specific inflammation. In constrictive pericarditis group 5 patients (42%) showed the presence of granulomatous inflammation suggestive of tuberculous infection and the rest (58%) showed nonspecific inflammation .In patients with cardiac tamponade only 2 (18%) were positive for tuberculous infection and the rest (82%) showed nonspecific inflammation (Fig : 21& 22)

Table 10: Treatment

	PE	CP	CT
Window Pericardiectomy	3 (11%)	Nil	11 (100%)
Treatment with ATT	3 (11%)	12 (100%)	11 (100%)

DISCUSSION

We analyzed totally fifty consecutive patients with chronic pericarditis. The principle aim of our study was to find the etiological profile in patients with chronic pericarditis. The study population were divided in to three major groups namely pericardial effusion; chronic constrictive pericarditis; cardiac Tamponade. 12 (24%) had constrictive pericarditis and 11 (22%) had Cardiac Tamponade and 27 patients (64%) had chronic pericardial effusion. Out of 27patients with chronic pericardial effusion 14(52%) patients had large pericardial effusion; 11(41%) patients had moderate pericardial effusion and 2(7%) patients had small pericardial effusion.

In our study population 50% were male. It was consistent with study by Sagrista-Sauleda J etal. He analyzed 322 patients with severe pericardial effusion. He found that 166 patients were (52%) men. The mean age of our study population was 34 ± 16.3. Our study population belonged to younger age group. In the study by Sagrista-Sauleda J etal the mean age of his study population was 56 +/- 17 years. His study population was predominantly in the elderly population. The mean ages of patients in three groups were 39, 20, and 34 respectively. The patients in constrictive pericarditis group were younger than the other two groups.

Dyspnea was the common presentation in all three groups in our study. In our study population only 4 patients (8%) had previous history of Tuberculosis. Clinical evidence of chronic renal failure was found in 4 patients (8%) and 6 (12%) patients had evidence of hypothyroidism.

The mean duration of clinical presentation prior to the study in all three groups was 4.8; 4.1; 3 months respectively. In the study by Chen KY, et al, the mean duration between the onset of symptoms and diagnosis of chronic pericardial effusion was longer in patients with shaggy-type effusion (39.6+/-8.7 vs. 21.0 +/-13.9 days, $p<0.05$).

In our study population pulsus paradoxus was found in 9 Patients (82%) in cardiac tamponade and 4 patients (15%) in severe pericardial effusion. In contrast, the study by Levine etal in patients with severe pericardial effusion with tamponade showed pulsus paradoxus in only 36% of patients. The mean Jugular Venous Pressure was 6.9; 7.5; 9.8 cm in the three groups respectively. In our study all patients with cardiac tamponade showed elevated Jugular Venous Pressure. In contrast, the study by Levine etal in patients with tamponade elevation of the jugular venous pressure was found in only 74%. In our study population 9 Patients (75%) in constrictive pericarditis group had pericardial knock.

In our study population 9 Patients (82%) with cardiac Tamponade had low voltage complex in ECG. In contrast, in study by Casale etal using Standard ECG criteria for low voltage (leads I, II, III each less than 0.5 mV, or V1 to V6 each less than 1.0 mV) , the low voltage complexes was found in 12% of patients with pericardial effusion with tamponade.

A thickened pericardium can be apparent in a simple chest x ray in case with pericardial calcification or in computed tomography or magnetic resonance imaging. In our study population 3 patients (25%) showed evidence of pericardial calcification. This is consistent

with other recent report. 27% incidence of pericardial calcification was reported in Being series and 5% incidence was reported by Cameron and colleagues in a series of 106 patients most of whom had idiopathic or post irradiation constrictive pericarditis. 53% incidence was reported in a more recent European cohort, in which idiopathic disease accounted for 50% of cases. An early report from the United States calcification was observed in approximately 50% of cases of constrictive pericarditis. A recent German study documented calcification in 55% of cases.

The mean pericardial thicknesses in the three groups were 2.29; 4.4 and 2.45mm respectively in our study. The mean MV flow velocity respiratory variation was high in patients with constrictive pericarditis. Amuthan v etal in his study of 16 patients with chronic constrictive pericarditis found, Doppler diagnosis of constrictive pericarditis was correctly made in 90% of patients. He used Doppler flow patterns of respiratory variation in ventricular filling (Mitral and Tricuspid forward flow) and central venous flow velocities (hepatic vein diastolic flow reversal) of more than 25% for the diagnosis of chronic constrictive pericarditis. Right atrial collapse and right ventricular collapse was found in all patients with cardiac tamponade. In the study by Levine and colleagues, 50 consecutive patients with severe pericardial effusion with tamponade were analyzed. Right atrial collapse was present in 92%, and right ventricular collapse in 57% of patients. In the study by Merce etal in 110 patients with moderate or severe pericardial effusion, only 34% of patients showed collapse of one or more cardiac chambers.

The mean amount of pericardial fluid in patients with cardiac tamponade was 650 ± 92ml. 6 patients (54%) in cardiac tamponade group had hemorrhagic pericardial effusion. The

mean pericardial thickness was high in patients with constrictive pericarditis.

DNA PCR for Mycobacterium Tuberculosis was done in 5 patients in the constrictive pericarditis group. Out of them 4 patients (80%) were found positive for tuberculous infection.

We found the most common etiology for all the three groups of chronic pericarditis was idiopathic. This was consistent with preexisting studies. In Patients with chronic constrictive pericarditis 7 patients (58%) belonged to idiopathic cause and in 5 patients (42%) it was due to tuberculous infection. Cacoub P et al studied the etiology profile in patients with constrictive pericarditis in 13 patients. He found neoplasia in 4 patients, pericarditis as a sequelae of radiotherapy in 2 patients, as a result of injuries in 2 patients. Pericarditis due to mediastinal and retroperitoneal fibrosis in 2 patients , post myocardial infarction pericarditis in 1 patient, purulent pericarditis in 1patient and bacteriologically proven tuberculosis in 1 patient.

Tuberculous infection was found in patients with cardiac Tamponade only in 2 (18%) patients.

In patients with chronic pericardial effusion specific etiology was found only in 13 (49%) patients. Out of them 6 patients (22%) were found to have hypothyroidism and 4 patients (15%) had pericardial effusion due to chronic renal failure. 1 patient (4%) had post Myocardial Infarction pericarditis; 1patient (4%) had pericardial effusion as a result of chronic Rheumatic carditis and 1patient (4%) had tuberculous etiology. The rest of the 14 (51%)

patients were idiopathic. In a study by Olivier Nugue etal, One hundred forty-one consecutive patients with unexplained pericardial effusion underwent pericardioscopy with a rigid mediastinoscope. For each patient, the etiologic data obtained by pericardioscopy (visualization of pericardium, guided biopsies, subxiphoid window biopsy, and fluid analysis) were compared with the results that would have been obtained with only conventional surgical drainage and biopsy (subxiphoid window biopsy and fluid analysis). After complete workup, a specific cause was found in 69 cases (48.6%); the other 73 cases were considered idiopathic effusions (51.4%). Colombo at al in his study he found that the etiologies of large pericardial effusion were: neoplastic (36%), idiopathic (32%), uremic (20%), post myocardial infarction (8%), and acute rheumatic fever (4%). Sagrista-Sauleda J etal in his study found that the most frequent etiologic diagnoses were idiopathic pericarditis (n = 66 [20%]), iatrogenic effusions (n = 50 [16%]), cancer (n = 43 [13%]), and chronic idiopathic pericardial effusion (n = 29 [9%]).

CONCLUSION

- In patients with chronic pericardial effusion specific etiology was found in 13 patients (49%). Out of them 6 patients (22%) had hypothyroidism; 4 patients (15%) had chronic renal failure; 1 patient (4%) had post Myocardial Infarction pericarditis; 1patient (4%) had chronic Rheumatic carditis and 1patient (4%) had tuberculous etiology.

- In CCP 5 patients (42%) had tuberculous etiology and the rest (58%) were idiopathic.

- In cardiac Tamponade 2 patients (18%) were found to have tuberculous infection and the remaining 9 patients (82%) were idiopathic.

- The most common cause in all the three groups of chronic pericarditis was idiopathic.

SUMMARY

We included fifty consecutive patients with features of chronic pericarditis in this study. The study was conducted in our department in Government Rajaji Hospital, Madurai. In our study 12 (24%) patients had chronic constrictive pericarditis (CCP) and 11 (22%) had Cardiac Tamponade (CT). 27 patients (64%) had pericardial effusion (PE). Out of 27patients 14(52%) patients had large pericardial effusion; 11(41%) patients had moderate pericardial effusion and 2(7%) patients had small pericardial effusion. Patients in Constrictive pericarditis group were younger age group than the others. The study population in constrictive pericarditis& cardiac Tamponade were predominantly male. In pericardial effusion group predominantly they were female. The mean duration of clinical presentation prior to the study was 4.8; 4.1; 3 months respectively. 16% of patients in the constrictive pericarditis group had previous evidence of Tuberculosis. 9 Patients (82%) in cardiac tamponade and 4(15%) in severe pericardial effusion group had pulsus paradoxus. The mean JVP was 6.9; 7.5; 9.8 in the three groups respectively. 9 Patients (75%) in constrictive pericarditis group had pericardial knock.

Most common etiology in all the three groups was idiopathic. In CCP 5 patients (42%) had tuberculous pericarditis and the rest (58%) were idiopathic. In patients with cardiac Tamponade 2 (18%) patients had tuberculous etiology and the rest (82%) were idiopathic. In patients with pericardial effusion 14 patients (51%) were idiopathic and 6 patients (22%) were hypothyroid and 4(15%) patients were in chronic renal failure. The rest of 3 patients belonged to Myocardial Infarction; Rheumatic fever and tuberculous etiology. The mean pericardial thicknesses in all the three groups were 2.29; 4.4 and 2.45mm. The mean MV flow velocity respiratory variation was high in patients with constrictive pericarditis. DNA PCR for

Mycobacterium Tuberculosis was positive in 4 patients (80%) in the constrictive pericarditis group. Pericardial biopsy was suggestive of tuberculous infection in 5 patients (48%) in constrictive pericarditis group and 2 patients (18%) in cardiac tamponade group.

We conclude that the most common cause in all the three groups of chronic pericarditis was idiopathic. Hypothyroidism as a cause for chronic pericardial effusion was found in 6 patients (22%). Chronic renal failure was found in 4 patients (15%) with chronic pericardial effusion. Only one patient (4%) in chronic pericardial effusion had tuberculous infection .In CCP 5 patients (48%) had tuberculous etiology and the rest (52%) were idiopathic. In cardiac Tamponade 2 patients (18%) were found to have tuberculous infection and the remaining 9 patients (82%) were idiopathic.

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PROFORMA

ETIO - PATHOLOGICAL PROFILE OF CHRONIC PERICARDITIS

NAME: **AGE:** **SEX:**

IPNO: **CDNO:** **ADD:**

1. Chest Pain Duration:
2. Breathlessness
3. Facial Puffiness And Leg Swelling
4. Fever
5. H/O TB
6. H/O Trauma/Surgery
7. H/O Rheumatic Fever
8. H/O Irradiation
9. H/O MI
10. H/O Renal Failure/Dialysis
11. H/O Drug Intake
12. H/O Hypothyroidism
13. Respiratory Rate
14. Pulse Rate
15. Blood Pressure Pulsus Paradoxsus:
16. JVP
17. Heart Sounds
18. Pericardial Knock
19. Pericardial Rub
20. Hepatosplenomegaly

INVESTIGATIONS:

1. **HEMOGRAM** HB TC DC N L M E B
2. **ESR**
3. **BLOOD SUGAR** Urea Creatinine
4. **CXR** CTR% Calcification RA LA RV LVApex AVGroove

ECG:

P	PR	Q	QRS	ST	T	QT/QTC	OTHERS
AMP Duran Axis			AMP Duran Axis				

ECHO: M MODE :

1. LVIDD : LVIDS : EF:
2. LA Size :
3. Aorta
4. Calcification
5. Septal Notch
6. Post Wall Movement.

2D ECHO:

		MED-LAT	SUP-INF
1.	LA (MM)		
2.	RA (MM)		
3.	Volume (CM^3)	RA	LA
4.	RVID (MM)		
5.	Septal Bounce		

DOPPLER :

1. Hepatic Flow
2. Pulmonary Flow
3. Mitral Flow

PERICARDIAL FLUID ANALYSIS :

Volume :	Colour :				
TC:	DC	N	L	E	M B
Protein	Sugar		LDH		ADA
Culture	Bacterial		Fungal		TB
DNA PCR					
Antibody					

PERICARDIAL BIOPSY

Histopathology:				
Culture	Bacterial	TB	Fungal	DNA PCR

MANAGEMENT :

MEDICAL

1. ATT
2. Steroids
3. NSAIDS

SURGICAL :

1. Window Pericardiectomy
2. Pericardial Resection

Balashankar